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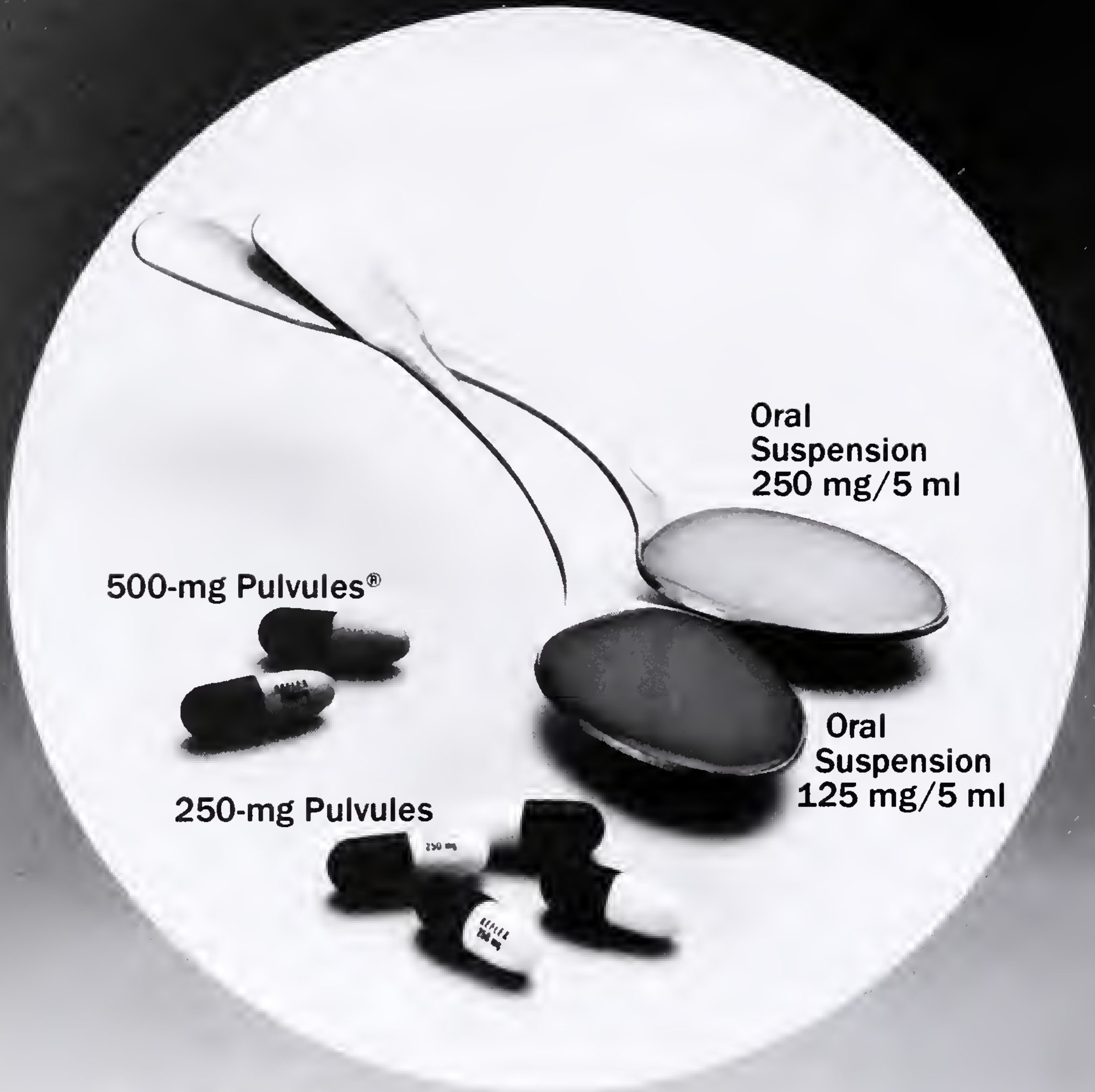

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FLEXIBLE FIBEROPTIC SIGMOIDOSCOPY

Wm. H. Bowers, MD, FACP, FACG

Introduction

Since 1975 flexible fiberoptic sigmoidoscopy (FFS) has been utilized increasingly by gastroenterologists, colorectal surgeons, internists, and family practitioners. There have been sundry sorts of 3-5 day courses offered to familiarize physicians who have not had experience with FFS in their training programs. Those courses have been reasonably successful with "hands on" participation.

There are several questions regarding FFS that should be addressed:

How much training and skill is necessary?
How difficult is it to learn?
What are the advantages and disadvantages?
What are the indications?
What is the cost effectiveness of FFS?

Unlike rigid sigmoidoscopy which is learned with little formal training in residency programs, FFS requires some instruction time. Various studies have shown that 6-8 FFS experiences render to the physician a reasonably adequate proficiency. However, 20 seems to make one more adept and adequate and 50 may result in a reasonably high degree of proficiency (1). In many instances a refresher lesson at 20 procedures is most helpful in ironing out minor difficulties.

Training and Skill

The neophyte doing FFS averages 11.4 minutes traversing 2-3 times the distance usually covered by rigid sigmoidoscopy. With proficiency the time may be cut to 6-8 minutes (2). Studies of family practice residency programs have shown that patients are more likely to be examined when FFS is used in the program (3). In training programs the entire 60cm of the scope is not always inserted. Some groups average only 55cm (a distance far better than the average 20cm for rigid sigmoidoscopy) (4). The shorter distance is traversed usually because of bowel spasm, patient discomfort, poor prep, and/or the skills of the neophyte flexible sigmoidoscopist. Inserting the sigmoidoscope rapidly while insuflating large quantities of air may reduce the distance traversed. Deflating the bowel allowing the colon to telescope over the scope remedies this mechanical problem.

Advantages and Disadvantages

Major disadvantages of FFS include, a) the breakdown problems resulting from a delicate instrument being handled or stored improperly, b) allowing cleaning solution or stool to dry in the channels, c) bending the scope in a greater than 8 inch diameter circle breaking fiber bundles, d) loosening or breaking cables, and e) getting liquid into a non submersible head. The initial capital outlay of $3000-$5000 as compared to $400 for a rigid scope often deters the solo practitioner but not groups or hospitals. The biopsy obtained by FFS is much smaller than with the rigid scope. This is a great disadvantage when one is trying to prove the existence of Crohn's colitis.

Advantages are: a) better patient acceptance especially when one compares Sim's position to the knee-chest, b) the distance traversed is 2-3 times that of rigid sigmoidoscopy with a greater yield of pathology [2-6 times as great for polyps, 2-3 times as great for cancer], c) there is a 75% reduction in patient discomfort with FFS, d) various photographic equipment such as low-cost Polaroid is available for documentation of lesions. Ninety four percent of residents prefer FFS when compared to rigid sigmoidoscopy where skills are never systematically taught nor competence measured. Biopsy techniques are easier with FFS than with rigid sigmoidoscopy and complications such as bacteremia and perforation are less frequent.

Indications and Costs Effectiveness

In a society where 5% of the population will suffer from colorectal carcinoma and where colorectal carcinoma is a major cause of premature death of 20-65 year olds, better screening is needed. FFS offers this.

Studies have shown 90% of women older than 40 have breast and pelvic examinations with PAP tests but only 42.7% have (rigid) sigmoidoscopies. Asymptomatic cervical carcinoma detection rates are 2.1 cases per 1000 exams. Applying statistical analysis in the same group of asymptomatic individuals who might undergo FFS would yield 74 adenomatous polyps and 8 carcinomas (5). FFS is truly a cost effective exam when one figures the medical costs of a Duke's A or B carcinoma allowed to advance to a Duke's C or D.

The cost of FFS to the patient has been reasonable as well. California Standard Nomenclature has coded 45330 for billing purposes of the "modified colonoscopy".

Examinations every three years after 2 initial negative examinations one year apart are recommended by the American Gastroenterological Association (AGA) in asymptomatic individuals over 40 years of age. The author's bias is every 1.5 years after 2 initial negative examinations one year apart in those over 35 years of age because human nature tends to make the patient delay to or beyond the recommendations of the AGA. The goal can only be accomplished if all physicians begin screening asymptomatic patients when they show for yearly exams. Only then can the annual 138,000 cases of newly diagnosed colorectal carcinoma and 1000 deaths/week be altered to any significant degree. This statistic has remained stationary for the last 25 years as has the overall 5 year survival of 41% (5).

Who should be screened if all individuals over 35 can't possibly be scoped? I would suggest that the following might have FFS; a) those having comprehensive examinations (excutive physicals etc.), b) those over 40 or 45, c) or anyone with a previous history of colon carcinoma or polyps (although complete colonoscopy is a much better surveilance), d) female patients with genital or breast cancer, e) those with a family history of colon, breast, cervical or pancreatic cancer, f) the presence of any of the following—radiation colitis; inflamatory bowel disease (complete colonoscopy is also better in these instances); diarrhea, constipation or other changes in bowel habits or caliber; low back pain, hematochezia; itching; anal fissures; anorectal fistulas or abscesses; rectal prolapse; a history of diverticular disease; hemorrhoids; gas; bloating; burning; weight loss; incontinence, soiling or abdominal pain all merit FFS (6).

FFS is useful for follow up after sigmoid or low anterior resection for carcinoma; after subtotal colectomy and ileoproctostomy for familial polyposis; for anastomotic reoccurence with rectal, rectosigmoid and obstructing sigmoid tumors; for investigations of stenosis secondary to perirectal inflamatory reactions; for functional bowel or decompression of sigmoid volvulus on an emergency basis before surgical correction.

A significant lesion detection rate of 39% is noted for FFS compared to only 13% for rigid sigmoidoscopy in some large studies (5). Other studies show 82% of polypoid lesions to be beyond 20cm, the usual distance traversed by rigid sigmoidoscopy. Statistics from 1939-1957 show the incidence of rectal carcinoma to be 51% and sigmoid carcinoma to be 13% in those having colorectal cancer. The numbers changed to 32% and 31% respectively from 1970-1977 (2). FFS offers a better chance to find higher lesions. No one knows why the change has occurred. Due to the increased incidence of higher lesions better and more extensive surveilance is mandated. *In 30% of patients symtomatic for neoplastic lesions there is no history of a rectal bleed or occult blood loss but only a change in bowel habits, anemia, weight loss, pain or a history of polyps (5).*

Preparation

Although some feel a single Fleets enema is adequate preparation, 2 Fleets given 15 minutes apart seem to yield the best results, especially if one wishes to utilize FFS on the initial visit to the office. It takes 30 seconds to administer each with 8 minutes retention before evacuation.

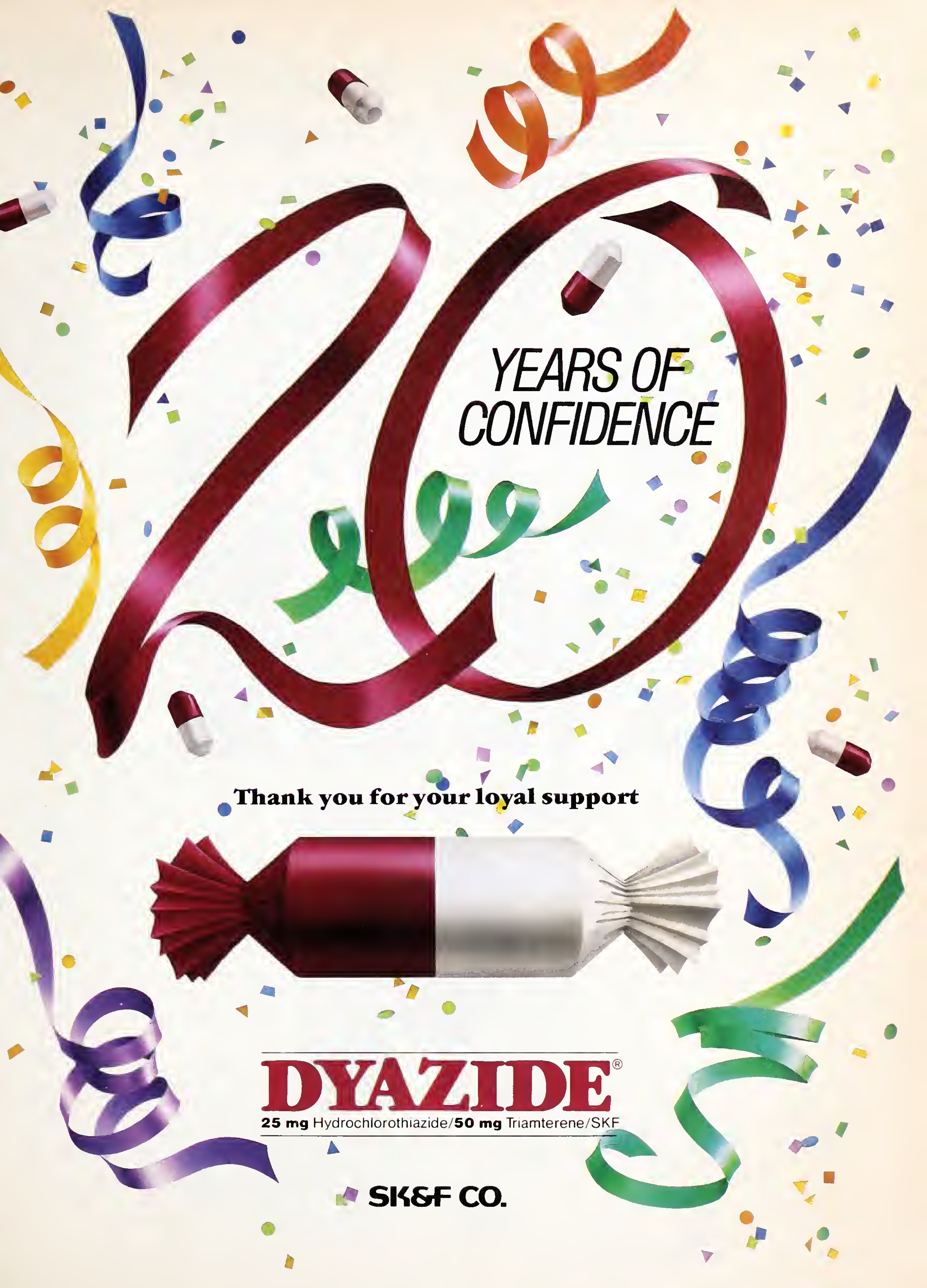
If the patient is kept NPO after midnight except for a clear liquid breakfast, two fleets enemas one hour apart two hours prior to the exam is adequate preparation to 60cm in 97% of the individuals. Stool coming down from above with the second enema is not really a problem. Placing the patient on a 2-3 day liquid diet as well as administration of GoLytely, magnesium citrate or ducolax seems to be overpreparation and adds to patient discomfort.

The procedure is best performed in the Sim's position affording maximum patient comfort and dignity. Nevertheless some physicians still prefer the knee-chest position with the patient on a tilt table.

Materials and Procedure

Besides the flexible sigmoidoscope, light source, biopsy forceps, cleaning brush (all of which come with the scope), a suction machine, basins for washing, betadine solution (providone iodine 10% generic is 1/3 the cost), alcohol and water are needed. A standard examination table is adequate. Even a gurney can be utilized.

When the procedure is about to be done a lubricating jelly is smeared on the distal end of the scope with the gloved hand taking care not to smear the lens at the tip. After performing a digital



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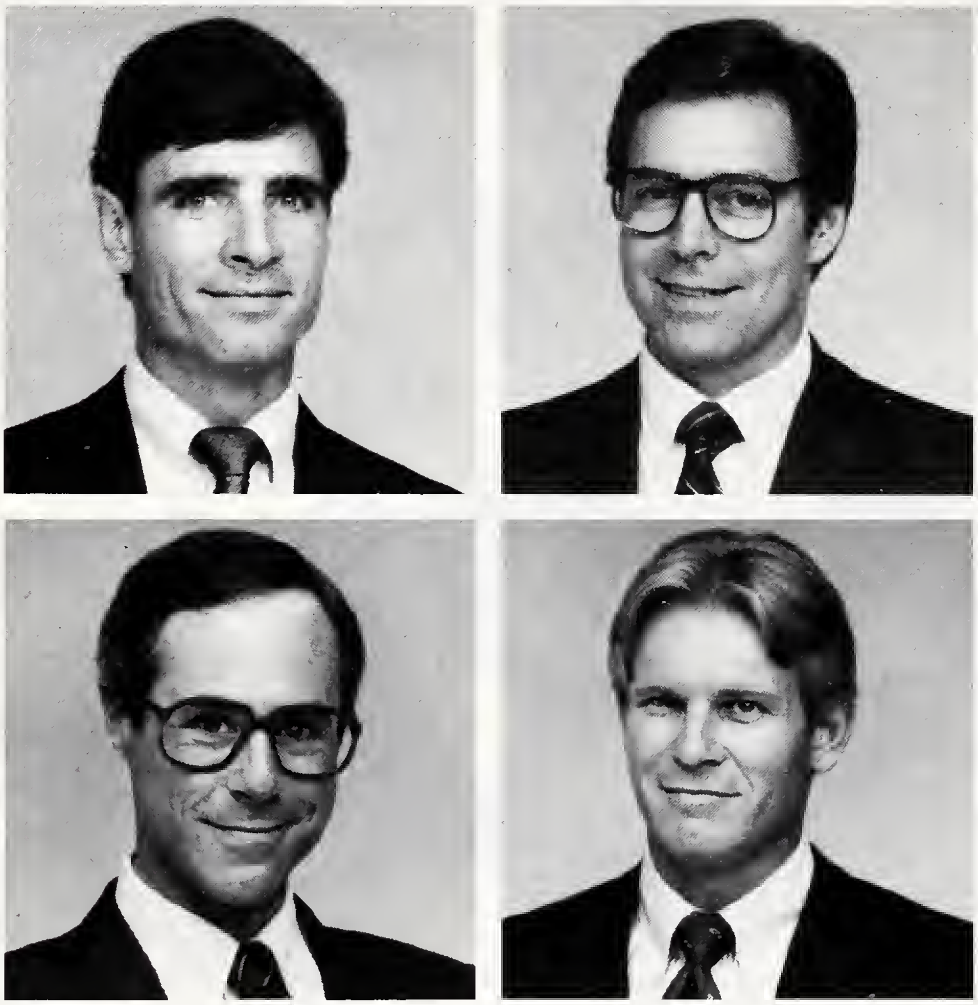

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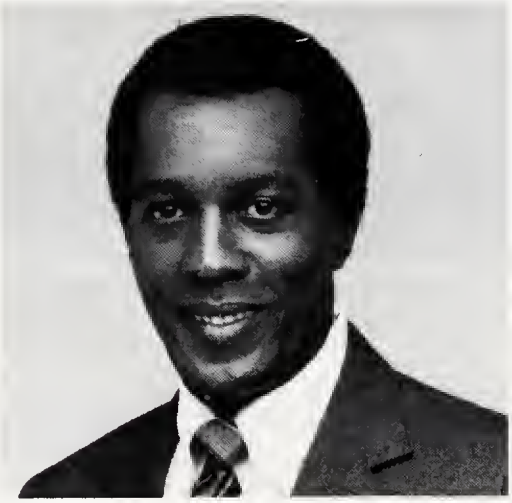

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rectal exam, the operator inserts the scope by grasping the tip with one hand, placing the index finger on the distal end, and inserting the scope tip into the anal canal in a sideways fashion. One should begin to insuflate air but be sure to inform the patient that he may experience the urge to defecate when the rectum is distended. The scope is advanced with lumen always in view. At 15-20cm a sharp angulation is reached. The scope is then advanced toward the concavity of the valves if lumen cannot be seen. This is the first blind spot one encounters. It is a place where a polyp or small tumor could be missed. It is usually well visualized upon exiting with careful circumferential inspection.

When in doubt, pull it out. Most neophytes as well as semi-experienced endoscopists don't withdraw when they can't advance. Believe me, it will save time in the long run as well as afford a complete exam.

As the scope is passed up the sigmoid an alpha shaped loop may be encountered. The small calibered flexible sigmoidoscope may be easily passed through the alpha loop but may not advance further. Withdrawing while rotating the scope conterclockwise on its axis (the alpha maneuver) will usually uncoil the sigmoid loop. Sometimes a reverse alpha maneuver, that is withdrawing rotating clockwise, is necessary if the loop twists in the opposite direction.

After the sigmoid is uncoiled the scope can easily be advanced up the descending colon to the splenic flexure. Often times with uncoiling of the sigmoid loop the distal transverse colon can be reached. It is recognized by the triangular luminal pattern.

When the scope has been inserted to the maximum it is slowly withdrawn carefully inspecting the bowel circumferentially. When the rectum is reached at 5-10cm the L-R and UP-DOWN controls are turned maximally and the scope is reinserted to 20cm. This is the J maneuver which allows inspection of the internal anal canal. The head of the scope is rotated clockwise then counterclockwise to allow circumferential viewing.

Not all physicians reach the 60cm mark but most can insert the scope to 55cm. Preventing factors may be spasm, patient discomfort, poor prep, or the skills of the new flexible sigmoidoscopist. Nevertheless, with time, practice and patience, FFS can become a most useful tool.

Cleaning

After the scope is soiled one should suction some cleansing solution up the scope to assure that no stool is clogging the biopsy channel. Wipe off any adherent material on the outside of the instrument. It is then placed in the basin with 500-700cc of solution consisting of 1/3 betadine, 1/3 alcohol, and 1/3 tap water. Make sure that the scope head doesn't get wet. A 2-5 minute soak is necessary. Remove the biopsy valve cover and unscrew the biopsy valve. Place them in the soaking solution and then pass the cleaning brush down the biopsy channel several times. The outside of the scope is washed off with several 4 × 4's soaked in cleaning solution. About half the soaking solution is then suctioned through the scope after the biopsy channel cleaning tube is attached. Another basin with tap water is used to rinse the scope and the outside is wiped with water soaked 4 × 4's. The scope is then wiped down with 4 × 4's soaked in 70% alcohol. An alcohol soaked Q-tip is used to clean out the biopsy channel at the head of the scope. The suction valve also needs periodic cleaning.

If biopsy forceps are used, rinse them under running hot water to loosen the biopsy jaws. Silicone spray may also be helpful in maintaining the forceps in an operable condition.

After the biopsy forceps are soiled they are placed in the cleaning solution then rinsed in the 70% alcohol solution. They are best dried by coiling and blotting with a towel to prevent rusting. They should be opened and closed to assure proper functioning. The cleaning brush should also be soaked in the cleaning solution and blotted dry after the 70% alcohol solution rinse. A toothbrush is most helpful in cleaning biopsy forceps as well as the cleaning brush.

Studies have shown that vigorous cleansing as described can render the scope and equipment free of bacteria, viruses and other contagious organisms. Nevertheless, in a practice where the scope is used frequently, periodic culturing of parts is advisable to assure continued proper disinfection.

Some individuals are proponents of a gluteraldehyde soak for 10 minutes. Unfortunately the gluteraldehyde may damage the lenses on the scope, irritate ones olfactory nerve, hands, and eyes.

After the scope is cleansed compressed air is blown through the channels to free the scope of remaining liquid. This need not be done if the scope is going to be used immediately for another procedure. The outside is wiped dry and the scope is ready for either re-use or storage by hanging in a purchased or fashioned rack. Endo Systems offers a nifty acrylic module for $155 which holds one scope and accessories.

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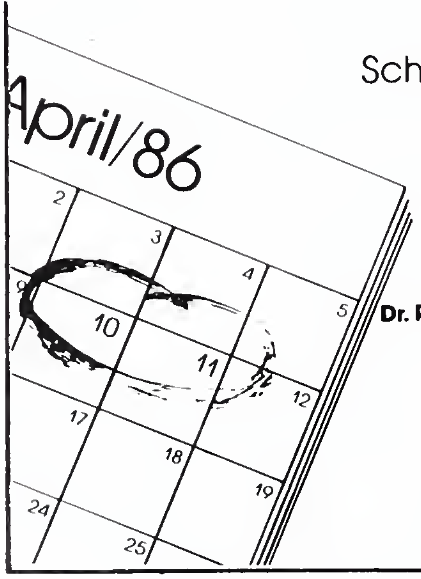

A VASOMOTOR COMPARISON OF THE HUMAN DIVING RESPONSE AND THE COLD PRESSOR TEST

Robert W. Arnold, B.S.

Abstract

The cold pressor test has been used clinically to induce vasoconstriction and hypertension. Although the diving response is also characterized by peripheral vasoconstriction, only its cardiac vago-tonic component has been used clinically in the treatment of paroxysmal supraventricular tachycardia. Five healthy adults underwent ice water hand immersion, and breath hold facial immersion at 1°C, 10°C and 25°C while electrocardiograph and venous occlusion plethysmography (contralateral arm) were monitored. A temperature dependent diving bradycardia was confirmed however cold pressor test did not alter heart rate. Apneic facial immersion produced a temperature dependent decrease in forearm blood flow with a maximum of 62% decrease in ice water. The contralateral forearm blood flow decrease due to ice water cold pressor test and facial immersion at 25°C were equivalent (22%). The diving response is therefore recommended as a non-pharmacologic vasoconstriction maneuver.

Introduction

Immersion of the hand in cold water (the cold pressor test), and immersion of the face in cold water (the diving response) are clinically useful physiological maneuvers which produce peripheral vasoconstriction. They share a common vasomotor component and a similar afferent receptor mechanism. The surface area of skin immersed in the cold pressor test and in the diving response are similar, and the thermosomesthetic afferents from the hand and from the face have a similar representation in the sensory homunculus of the post-central gyrus. The propensity for these tests to elicit peripheral vasoconstriction has not previously been compared.

The diving response is composed not only of vasoconstriction but also of a vagal bradycardia which, in humans, is markedly temperature dependent (1,2). Though not explicitly stated, data from some studies suggest a temperature dependence of the vasomotor component of the human diving response, also (2,3). The following study investigates the temperature dependence of forearm blood flow during the human diving response in comparison to the contralateral forearm blood flow response with the cold pressor test.

Methods

Five healthy subjects, four male and one female, (aged 24-42) signed an informed consent under the approval of the Yale University Committee on Human Investigation.

The diving response was elicited by seated, 30-second, mid-inspiratory breath hold facial immersion in a basin of water. Subjects were instructed to avoid Valsalva maneuver during breath hold. The diving response for each subject was tested at three water temperatures: 1 ± 1°C, 10 ± 1°C, and 25 ± 1°C. The cold pressor test was elicited by 30-second, eupneic immersion of the right hand to the level of the wrist in a second basin of ice water (1 ± 1°C) located on a bench to the right of the subject. The diving response at each temperature and the cold pressor test were repeated three times in random order for each subject. A recovery period of at least two minutes separate each maneuver.

Heart rate was extracted from R-R intervals on a continuous, calibrated electrocardiograph (Hewlett-Packard 1500B). A pre-dive heart rate average was obtained from 3 to 15 seconds before immersion to exclude the pre-dive tachycardia frequently associated with the last breath (3). A dive heart rate was averaged from 15 to 30 seconds of immersion.

Forearm blood flow of the left (contralateral) arm was obtained by mercury-in-silastic strain gauge, venous occlusion plethysmography using a modified Whitney technique (4,5). The pre-dive forearm blood flow was measured 15 seconds before immersion and the dive value was obtained after fifteen seconds of immersion. The term "dive" is used to describe immersion of either the face or the hand in this paper.

Because of the characteristic individual variability of the human diving response (6,7), heart rate and forearm blood flow are presented as relative changes and data are analyzed by two-way analysis of variance (ANOVA).

Results: Figure 1 shows heart rate and Figure 2 shows forearm blood flow responses of five subjects for facial immersion at three water temperatures and for ice water cold pressor test.

Heart Rate: The temperature dependence of human diving bradycardia was confirmed:

Water Temperature	%Decrease in Heart Rate
25°C	8%
10°C	12%
1°C	34%

The cold pressor test produced no change in heart rate.

Forearm Blood Flow: Two-way ANOVA revealed minimal intra-subject variation in forearm blood flow response $\{F_{4,8} = 1.2, p \blacktriangleright .05\}$ but a highly significant temperature dependence of dive forearm blood flow $\{F_{2,8} = 12, p \blacktriangleleft .01\}$:

Water Temperature	% Decrease in Forearm Blood Flow
1°C	62%
10°C	40%
25°C	21%
CPT	22%

If the dive forearm blood flow is expressed as a percentage of pre-dive forearm blood flow (FBF%), then the temperature dependent diving forearm blood flow significantly ($r^2 = 0.65$) fits the following regression:

$$FBF\% = 1.5(^{\circ}C) + 41\%$$

Using this relationship, the 22% forearm blood flow decrease during contralateral ice water cold pressor test is equivalent to the human diving response at 24°C.

Discussion:

The cold pressor test (CPT), i.e. immersion of one hand in ice water to above the wrist, produces a non-pharmacologic adrenergic peripheral vasoconstriction and an elevation in blood pressure. The cold pressor test in 99% of subjects produces an average rise in blood pressure of 18-20 torr (8,9). The maximum blood pressure during the cold pressor test occurs after 20-30 seconds of immersion (10). The cold pressor test and isometric handgrip exercise produce similar increases in blood pressure but isometric handgrip produces more of an increase in heart rate. Immersion of the foot in 25°C water produces a decrease in forearm blood flow (3). Left ventricular end diastolic pressure and the rate-pressure product rise during the cold pressor test (11) but the effect on heart rate is either an insignificant change (3,8) or a moderate increase (9,12). The cold pressor test decreases contralateral forearm blood flow and was shown to increase total peripheral resistance from 1414 to 1686 dynes*sec*cm^{-5} (12). When the vasoconstriction of the cold pressor test is blocked by intra-arterial alpha-adrenergic antagonists (guanethedine or phentolamine) immersion of the hand in cold water produces a vasodilator component which can be partially blocked by atropine (13).

Clinically, the cold pressor test has found merit as a long term and short term vasomotor diagnostic maneuver. In 1934, 300 Rochester, Minnesota school children were screened for their blood pressure responsiveness to immersion of a hand in ice water (10). One hundred fifty one of these were restudied in 1961 (14). Although initally doubted (15), Wood, et al, demonstrated that hyperractivity to the cold pressor test in some of the 1934 students was a positive predictor of hypertension in 1979 (16). As a provocative pressor test in the diagnosis of ischemic coronary artery disease, the cold pressor test was favored over dynamic and isomeric exercise because of its smaller rate-pressure product (17). During cardiac catheterization in patients with coronary artery disease, the cold pressor test has been used to provoke a 27% increase in coronary vascular resistance, and this vasoconstriction can be reduced with phentolamine (18). Focal coronary spasm has been documented with cold pressor test during cardiac catheterization in patients with atypical angina or other chest pain syndromes (8,11). The cold pressor test is also used before glucagon stimulation test in the diagnostic work-up of pheochromocytoma (19).

Immersion of the face with or without breath hold in cold water is called the diving response. The human diving response is characterized by peripheral vasoconstriction, vagal bradycardia and maintained central mean arterial pressure (20). The diving response is dramatically temperature dependent so that colder water temperatures produce greater bradycardia (2). The diving response has been successfully applied clinically to treat paroxysmal supraventricular tachycardia in infants, children, and adults (21-25). It has also been used as a brainstem neurological test (26).

The cold pressor test has frequently found clinical utility to invoke vasoconstriction. The diving response has been successfully and safely used to treat paroxysmal supraventricular tachycardia because of its vago-tonic component. The diving response has received little clinical attention as a pressor, however.

While ice water cold pressor test will produce substantial vasoconstriction, as measured by contralateral forearm blood flow, breath hold facial immersion in 24°C water will produce an equivalent reduction in forearm blood flow. Facial immersion in water colder than 24°C produces even more vasoconstriction than ice water hand immersion. The percent reduction in forearm blood flow attributable to immersion of the face in ice water is three times that of ice water hand immersion.

The facial trigeminal cold receptors and nocioreceptors not only are afferents to a unique cardiac chrontropic response (diving bradycardia) but also trigger a temperature dependent vasomotor response which is stronger than can be elicited by stimulating similar receptors in the hand. Therefore, the diving response should be considered by the physiologist or clinician seeking a non-pharmacologic pressor stronger than the cold pressor test.

Acknowledgement: The facilities and advice of Ethan Nadel, Ph.D. are appreciated.

Figure 1

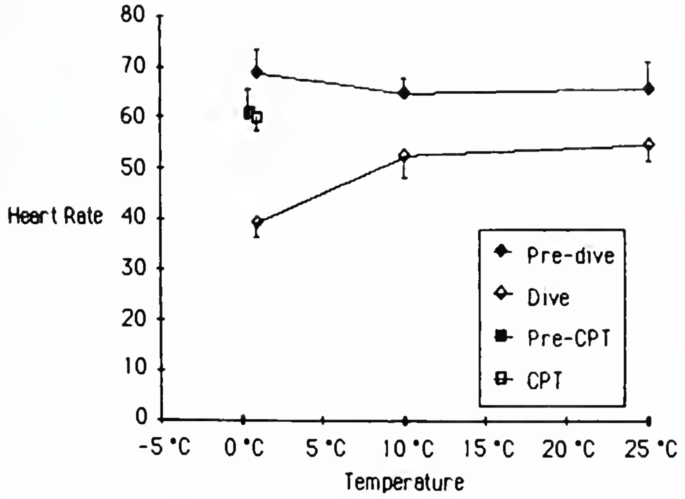

Figure 2

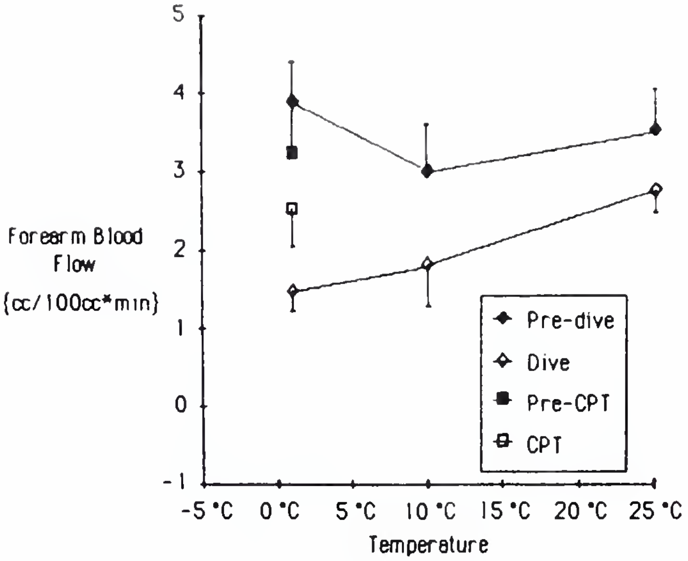

Figure Legends:

Mean ± S.E.M. HEART RATE for the diving response at 1°C, 10°C and 25°C (diamonds) and the cold pressor test (CPT) at 1°C (squares). Solid markers indicate pre-maneuver and open markers indicate maneuver values. n=5.

Mean ± S.E.M. FOREARM BLOOD FLOW for the diving response at 1°C, 10°C and 25°C (diamonds) and the cold pressor test (CPT) at 1°C (squares).

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ANTIDEPRESSANT DRUGS AND ALCOHOL AMNESIA

Charles J. Hudson, M.D.

Sherrod H. Perkins, M.S., C.S.C.

Abstract

Four clinical cases are discussed in which the individuals involved drank alcohol while on antidepressant medication and experienced amnesic episodes.

Since the initial report of alcoholics' blackouts associated with tricyclic antidepressants (1), the authors have interviewed four more patients who experienced their first blackout while on psychotropic medications. While not as uniform as the original group in which all three individuals experienced their blackout within a month after starting tricyclic antidepressants, these cases should alert other investigators and clinicians to a phenomenon which might be more extensive than is realized. The following case reports are part of a second series that the authors have experienced in clinical practice since the first paper was written.

Case I

The first case is that of a 26 year old single woman who presented to the psychiatric clinic with complaints of mood swings during which she might misuse alcohol, lose control of her behavior, and become verbally abusive to individuals to whom she was close. In between these episodes she drank socially in an acceptable way, as verified by her boyfriend. She further stated that her mother had been a bipolar manicdepressive and had been helped by taking lithium carbonate. She therefore requested lithium therapy for herself. The request seemed reasonable since her history could be compatible with a bipolar disorder, and she was begun on lithium carbonate and brought to therapeutic blood levels. She was asked to abstain from alcohol completely. She was able to do this initially, but over the ensuing weeks she began drinking again. She went to a party 10 weeks after starting lithium, drank alcohol, and experienced a blackout during the course of the evening. She was quite vivid in her description of loss of memory of the evening, and was certain that she had not had any prior, similar experience. What was most startling for her was being informed the next day that she "had sexual intercourse with three men in a field" and had no recollection of it. She denied having been intoxicated prior to blacking out.

Case II

A 46-year-old auto mechanic and used car salesman suffered a head injury when a car door was blown by the wind and hit him in the face, stunning him and causing loss of an eye. In the months following the injury he became depressed and sought psychiatric help. His mood disorder, diagnosed as a depressive neurosis, responded well to nortriptyline. He was able to resume work. He drank socially on weekends and denied ever having had any problems such as blackout, driving infractions, or any disruption in his personal life. This was confirmed by his wife. He was cautioned about using alcohol while on antidepressants, but he continued to drink "moderately" on weekends. On one of these occasions he blacked out for five to six hours and recovered his memory sitting in his truck in a parking lot several blocks from his home at 2:00 a.m. He had been on nortriptyline a year at this time.

Case III

A 43-year-old married man was interviewed at a regional mental health clinic, and assessed for counseling by an alcohol counselor. He had also been on medication prescribed by psychiatrists. He was hospitalized in 1984 for a psychotic disorder with alcohol abuse. He had been taking 200 mg amitriptyline a day for approximately a year and had remained alcohol free. He held a responsible job at a nearby college, but in the summer of 1984, while still taking antidepressants, he resumed drinking distilled spirits every day after coming home from work. He was quite consistent in saying that he had only mixed drinks before supper. He was cautioned on several occasions by the authors for two reasons: the first, for resuming alcohol with a history of alcohol abuse, and the second, for drinking while taking medication. In October, 1984, he informed the authors of the following episode. He came home from work as usual one day, had his drinks at supper, then settled down to watch television for the evening with his wife. The next day at work, he suddenly realized that he remembered nothing after 9:00 p.m. the night before. He did not recall how the television program ended, and did not recall going to bed. His wife related to him that he had seemed quite normal during the course of the previous evening and she had not been aware of any problem. She stated that when the television program was over they had gone to bed and he had taken his medication as usual. He had a similar experience two weeks later.

Case IV

A 44-year-old married woman had been on imipramine for seven years. She had a diagnosis of depressive neurosis (DMS-III), and had a history of social drinking, but indicated that she never had any major difficulties which could be characterized as alcohol abuse. On one night, after drinking a liqueur and some brandy, she remembers returning home but does not remember waking up her daughter and shouting, "I'm going to kill him!", referring to her husband, nor did she remember sleeping on the couch the remainder of the night. It should be noted at this point that her marriage had been a very stable, close, loving one, and thus her statement that she wanted to kill her husband was quite inappropriate, and she could give no reason for it.

Discussion

Several points seem relevant in considering the two series of cases which now total seven individuals. There are two men in the recent group, so it appears that this phenomenon is not limited to women. Three of the four present individuals had been on tricyclics for at least a year, unlike the original three cases who had experienced blackouts in their first month after starting a tricyclic. The occurrence of one woman who was taking lithium also is new.

Physiologically, it appears that alcohol and antidepressant drugs can trigger blackouts in some situations when they are both taken together. The biochemical mechanisms of mammalian memory storage are still far from being fully understood despite new hypotheses and research (2,3). Kandel's discussion of conditioning with invertebrate mollusks indicates how complex, memory processes may be even in lower forms of animals (4). At this point memory formation is a complex multi-staged process, comparable to blood clotting, so that interference in any one step can result in a final, common, clinical problem, i.e. bleeding in the case of hemostasis or a blackout when some aspect of memory formation is disrupted.

Epidemiologically, we do not know how common these blackouts are in persons taking mood-altering drugs, but we will continue to urge all of our patients on these agents not to drink, with a very clear explanation why. Another troublesome aspect of these blackouts is that it has not always been apparent to other people that the amnesic individual was in a crisis and needed supervision. In fact, one person drove while blacked out, another made verbal threats, and a third engaged in unusual sexual behaviors normally not characteristic of her.

I would like to thank Dr. Robert Sioussat for reviewing the manuscript and making a number of constructive comments.

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CORRECTION

Vol. 24, No. 4, Page 101 Footnote should read; * President, Union of American Physicians & Dentists, 1730 Franklin St., Suite 200, Oakland, CA 94612, (415) 839-0193.

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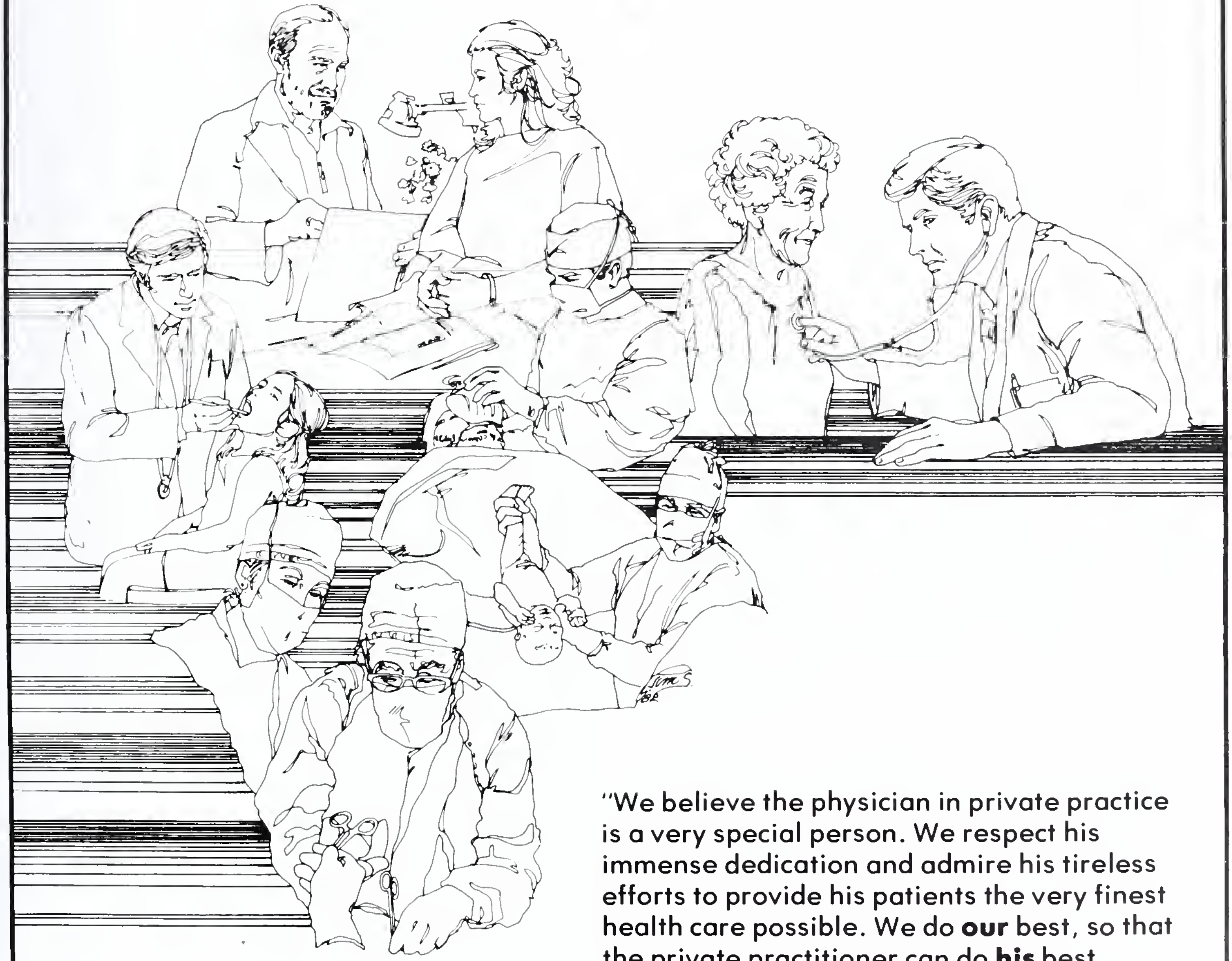

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NIFEDIPINE—INDUCED HEPATITIS

H. Gilbert Welch, MD

Burton Lazar, MD

Jacklyn Gresser, RN, BS

Brian J. McMahon, MD

Calcium channel blocking agents are regularly being utilized for a wide variety of cardiovascular disorders.[1] Certain adverse effects are well known and are primarily a consequence of the vasodilation and increased atrio-ventricular block that are also responsible for their therapeutic effect. Marked transaminase elevations are well described in the case of verapamil[2], consequently its manufacturer recommends periodic monitoring of liver enzymes.[3] In the case of nifedipine the picture is not as clear. The manufacturer lists mild to moderate elevations of transaminases as a rare side effect and notes a single incident of significantly elevated transaminases in a patient with a history of gall bladder disease after 11 months of therapy.[4] Two cases out of Great Britain report mild transaminase elevations after nifedipine administration, although subsequent biopsy of one case showed alcoholic liver disease.[5,6] We report a significant elevation in both SGOT and SGPT in a patient without preexisting gall bladder or alcoholic liver disease.

Report of a Case—A 50-year-old Eskimo woman was admitted to the hosptial with a three hour history of substernal chest pain, diaphoresis, and shortness of breath. She had been previously healthy and had not been admitted to the hospital for over a decade. She was under treatment for mild hypertension with a regime of hydrochlorothiazide (50mgs.) daily. She was on no other medications and had no prior history of liver or gall bladder disease. She denied any alcohol intake. Physical exam disclosed an obese woman in no distress. There was neither hepatoslenomegaly nor signs of hepatic failure.

Admission EKG showed no acute changes from prior studies. Laboratory studies disclosed the following values: Creatine Phosphokinase (CPK) 303 IU/L (reference range, 15 to 228 IU/L); Serum Aspartate Transaminase (SGOT) 37 IU/L (reference range, 6 to 22 IU/L); and Lactic Dehydrogenase (LDH) 218 IU/L (reference range, 113 to 246 IU/L). On her second hospital day the EKG had T-wave inversions in the anteroseptal, anterior, and anterolateral leads. These global changes of ischemia without associated Q-waves led to the diagnosis of subendocardial myocardial infarction. Cardiac enzymes peaked on Day 2 with a CPK of 480 IU/L; SGOT of 61 IU/L; and a LDH of 274 IU/L. The patient was mildly hypertensive and was started on propranolol 10 mgs. TID and nitropaste 1" QID.

On hospital Day 4 measured CPK was 84 IU/L, SGOT 31 IU/L, and LDH 200 IU/L. She began to have intermittent chest pain and was started on nifedipine 10 mgs. TID on Day 5. On Day 6 the SGOT was 63 IU/L, rose to 205 IU/L on Day 7, and peaked at 361 IU/L on Day 8. There was no concomitant rise in CPK. On Day 9 the LDH was 371 IU/L, Serum alanine transaminase (SGPT) 426 IU/L (reference range, 2 to 33 IU/L), alkaline phosphatase 137 IU/L (reference range, 23 to 98 IU/L); and a total bilirubin less than 1 mg/dl. The elevation of both transaminases with no change of CPK was felt to represent hepatic insult. The patient felt well and her physical exam remained unchanged. Subsequent information included a positive hepatitis A IgG antibody with a negative IgM antibody. The patient was also positive for hepatitis B core antibody, but was negative for surface antigen and antibody as well as IgM anti-core antibody. These were indicative of remote hepatitis A and B infections.

The addition of nifedipine to the patient's regime was felt to be the cause of the chemical hepatitis. Consequently, it was stopped on the tenth hospital day. On Day 12 the SGOT had diminished to 44 IU/L and the SGPT to 172 IU/L. At three weeks the transaminases had normalized to a SGOT of 34 IU/L and SGPT 36 IU/L. At this time the patient was started on diltiazam without any subsequent enzyme elevations. Since the patient had suffered a recent myocardial infarction, it was decided not to do a liver biopsy or a rechallange of nifedipine.

Comment—A review of the literature reveals that hepatocellular injury has been reported previously with nifedipine, although not to the extent described here. The drug is believed to be rapidly and completely metabolized by the liver to inactive metabolites[7,8], thereby placing the organ at risk for a hypersensitivity reaction. This is the proposed mechanism for the hepatitis described by Rotmensch et al[9] although the transaminase elevation was relatively mild (SGOT 56 IU/L and SGPT 68 IU/L). In that case production of migration inhibition factor against nifedipine indicated that the patients lymphocytes had become sensitized towards the drug, suggesting an allergic mechanism. The FDA reports 27 cases of liver function abnormalities associated with nifedipine and one case where the patient had been left on the agent with persistently elevated transaminases and remains asymptomatic.[10] The reports received by the FDA do not include any irreversible hepatic injuries.

The clinical features of this patient also suggest an allergic mechanism. Although never rechallenged with nifedipine, the temporal course of the transaminase elevation (see figure 1), in the absence of other medication changes, and the results of the viral hepatitis panels strongly suggest nifedipine as the causative agent. The rapid improvement in the biochemical abnormalities with the discontinuation of the agent point to a hypersensitivity reaction. The potential for hepatocellular injury albeit rare, ought to add nifedipine to the list of calcium antagonists whose use warrants periodic monitoring of liver enzymes.

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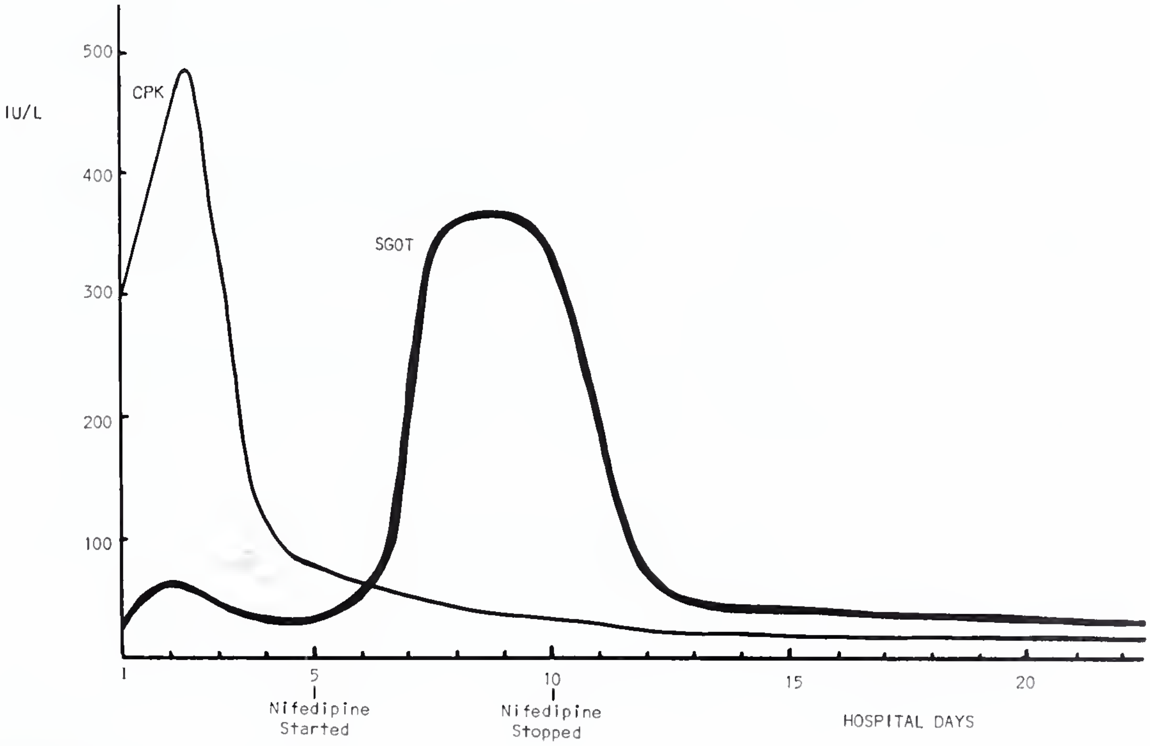

Figure 1: Temporal Course of CPK and SGOT

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DIPHTHERIA AND ALASKA

Diphtheria possibly was described by Hippocrates. However, there is no other record of it until the late 16th century. Diphtheria, thus historically, is a post-Renaissance sickness.

It is the epidemic aspect of the disease which has set it apart. In 1735, there was an outbreak in New England which lasted for five years. At the same time, there were similar epidemics in England, France and the West Indies. After this, the disease appeared approximately every 25 years. It seemed to gather in virulence, culmulating in a dreadful epidemic in 1857 in Europe and America.

Diphtheria had become a scourge just as scientific medicine had begun to emerge.

Just over 100 years ago in Germany, Friedrich Loeffler published a monograph in which he proved that a particular bacterium caused diphtheria. Four years later, Pierre Paul Emil Roux demonstrated that the bacillus produced a toxin. It was the toxin which caused paralysis and heart failure. It was Emil von Behring in Germany in 1895 who produced an antitoxin, for which he was to receive the first Nobel Prize for medicine.

Diphtheria occurs mainly in children. It has been referred to as a malignant sore throat. A grey, green or white membrane covers the throat: Death comes by suffocation, heart failure or paralysis.

The first therapy attempted to relieve suffocation. Tracheostomy was performed as early as 1825, but only a few survived the disease and the operation. In children the mortality was 80 per cent.

Joseph O'Dwyer, in 1895 in New York, successfully treated diphtheria with intubation. Because of this, the survival rate began to rise slowly.

In 1924, diphtheria toxoid was prepared for immunization. Wade Hampton Frost, at the Johns Hopkins School of Hygiene and Public Health, demonstrated that this immunization worked in school children.* In 1928, Baltimore began an active immunization program.

The control of diphtheria, an international effort, was at hand just as the most dramatic outbreak started in Nome, Alaska.

In 1925 there were 1429 living in Nome. However, there were 10,000 more island and coastal people who looked upon Nome as the center of civilization. Nome was then served by a lone missionary doctor, Curtis Welch, at the 25 bed Maynard-Columbus Hospital.

When Dr. Welch found diphtheria in the community in January 1925, he had on hand 75,000 units of five-year-old antitoxin. The Eskimos had little or no immunity to white man's diseases. Nome was immediately quarantined to try to prevent spread, the outdated antitoxin was put to use, and an appeal was sent out for help. By January 31 there were 22 definite victims and 20 suspected cases. Five had died.

The U.S. Army Signal Corps had a network of wireless and telegraph stations which relayed the news to the rest of the state, the nation and the world. The U.S. Public Health Service collected 1,100,000 units of antitoxin and routed it to Seattle for shipment to Seward. Meanwhile there were 300,000 units in Anchorage. Dr. J.B. Beeson, the railroad physician, placed this as a 20-pound package on the train bound north to Nenana.

Northern Commercial had trading posts which crossed Alaska and were used as stations to forward mail from Fairbanks to Unalakleet. From November to May sled-dog teams carried this mail. Using this network as a basis, word passed by "mukluk telegram" to the villages where a collection of the best dogs and mushers to supplement the regular runners were selected.

The mushers were Eskimo, Russian-Eskimo, Norwegian, Irish and Koyukuk and Yukon Indians. This is how they ran from Nenana:[1]

1. "Wild Bill" Shannon	Jan 27-28	To Tolvana	52 mi
2. Dan Green	28	To Manley Hot Springs	31 mi
3. Johnny Folger	28	To Fish Lake	28 mi
4. Sam Joseph	29	To Tanana	26 mi
5. Titus Nicholi	29	To Kallands	34 mi
6. Dave Corning	29	To Nine Mile	24 mi
7. Edgar Kalland	29	To Kokrines	30 mi
8. Harry Pitka	29	To Ruby	30 mi
9. Bill McCarty	29	To Whiskey Creek	28 mi
10. Edgar Nollner	29-30	To Galena	24 mi
11. George Nollner	30	To Bishop Mt.	18 mi
12. Charles Evans	30	To Nulato	30 mi

Fifty-four hours after the start at Nenana, the serum reached the Idiatrod Trail at Nulato.

13. Tommy Pasty	30	To Kaltag	36 mi
14. Jackscrew	30	To Old Woman	40 mi
15. Victor Anagick	30-31	To Unalakleet	34 mi
16. Myles Gonangnan	31	To Shaktolik	40 mi
17. Henry Ivanoof	31		
18. Leonhard Seppala	31-Feb 1	To Golovin	91 mi
19. Charles Olson	1	To Bluff	25 mi
20. Gunnar Kaasen	1-2	To Nome	53 mi

---

*As a small child in Baltimore, I was discovered to be a diphtheria carrier during Dr. Frost's work and was quarantined at home for six weeks. I would have felt better at the time had I known I was to be a particle of history.

1. Modified from a chart in Ungerman: The Race To Nome.

Sam Joseph and Harry Pitka, both with seven dogs, averaged nine miles per hour; Tommy Pasty, with good conditions, averaged better than ten miles per hour.

Henry Ivanoof had barely left Shaktolik when he met Leonhard Seppala. Seppala had the only Siberian Husky team, known for its speed. He had taken a double team from Nome: one rode; the other pulled. He had expected to go all the way to Nulato and back to Nome not knowing of the additional relay teams. He had already come 50 miles but turned back.

His lead dog, Togo, was twelve years old but safely led the team over the windswept ice of Norton Sound. While they eventually stopped overnight, the dogs had gone 84 miles without rest.

Charles Olson drove the toughest section of the trail. It was cold and windy. He had frost bite and two dogs had frozen groins.

Gunnar Kaasen was to have been relieved at Pt. Safety by Ed Rohn. There is controversy as to why he did not stop. He claimed he drove through blinded by snow. He had thirteen dogs led by Balto. His sled tipped over, and he lost the serum briefly. Balto led the team in the darkness by instinct to Nome.

Billy McCarty at this writing, lives in Ruby; Charles Evans and Edgar Nollner in Galena.

Six hundred and seventy four miles of ice and snow covered terrain was traversed in 127½ hours. The serum run was accomplished in five and one-half days to the 25 usual mail service time.

Through wireless and the newspapers, the world followed the serum race. It was on front pages everywhere dominating other news. During the late 1920's, there must have been tens of thousands of dogs named Togo or Balto.*

Five days after the delivery, the second batch of 1,100,000 arrived in Seward. It was transported by special train to dog team at Nenana. There were many new drivers on this relay. They have never been recognized. Some of the original mushers ran but in different sections. They drove the 465 miles to Unalakleet in 80 hours. From there to Nome, records are unknown.

The quarantine was lifted in Nome on February 21.

The importance of this race can not be over estimated. It demonstrated sensationally the value of diphtheria antitoxin. Immunization became widespread around the world.

A final touch is that it prompted air mail service. Thought had been given to flying the serum to Nome, but it was not feasible. However, planning was now begun. This bred the famed Alaskan bush pilot.

In the next fifty years there were no notable diphtheria outbreaks in Alaska. Then true to form, diphtheria reemerged. On July 23, 1975, a woman died of diptheria in Anchorage despite the use of antitoxin, penicillin and tracheostomy. She suffered both cardiac and neurologic symptoms.

In 1975-76, there were twelve cases of diphtheria, including a cook in the pipeline camp at Gakona.

Because school children are required by law to be immunized, diphtheria has become an adult disease. Diphtheria bacteria are carried in the human throat. Since carriers are asymtomatic, the constant moving about of the latter twentieth century population exposes the unimmunized to this disease. There were eleven carriers in Gakona. Another carrier was an airline stewardess.

A vast immunization was organized to give diptheria-tetanus (Td) booster injections. This was begun in Palmer and Wasilla. Appropriately, one of the clinics was set up at the Iditarod School in Wasilla.

A massive effort was made in Anchorage, reaching out to the whole state. Twenty-five thousand dollars was spent, small money, considering the ugly possibilities. Enormous effort was made by volunteers. Nurses and physicians freely gave their services. Those who came for injections often stayed to unpackage syringes and toxoid to speed up the process. Air guns were provided by the Communicable Disease Center in Atlanta, Ga. so that the individual had a choice of method of immunization. Afterwards, it was discovered that the air jets caused more side effects than the needles.

Diphtheria occured in Soldotna, Seward and Anchorage. For a time, the oil platform Dolly Varden in Cook Inlet was quarantined, but the patient proved to have a sore throat not due to diphtheria.

By November 19, 1975, 183,740 people had been immunized in the state in public clinics. The number who got immunization in private clinics is not known. In addition, there were the school children who had current immunization. Two thirds of Alaska was thus known to be protected.

When Dr. Roux carried out the first large scale clinical trials in Paris, 1893-94, he noted that the case fatality ratio even with antitoxin was ten per cent. This has remained constant for over 90 years. This means that even with modern care and antibiotics ten per cent of diphtheria victims die.

Also, it is prudent for those recovered from diphtheria to be immunized since it can not be shown that having had the disease provides future immunity. Immunization only protects against the toxin which causes cardiac and neurological complications. It does not prevent infection by the organism of throat or skin. Individuals can still carry the bacteria or have pharyngitis even though immunized.

Alaska can be proud of its accomplishments because of progressive immunization and public health practices. In 1974, no cases of measles or rubella were reported. Alaska led the nation in being the first state to free itself for an entire year of a communicable disease.

In 1977, Alaska became the first state rigouously to enforce school immunization requirements.

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* One of which was my husband's family.

Now it seeks to be the first state to institute a program to vaccinate adults against tetanus and diphtheria every ten years.

The Alaska Department of Health and Social Services has initiated another massive campaign to vaccinate all Alaskans who have not had tetanus-diphtheria immunization within the past ten years. The Municipality of Anchorage Department of Health and Human Services will operate the ten year booster campaign in Anchorage. The rest of the state will be covered by the Communicable Diseases Section of the Alaska Department of Health and Social Services.

The state will provide vaccine free. Beginning January 1, 1986, all physicians will be urged to immunize their patients. During the next three months effort will be made to publicize the program widely, to facilitate a campaign for booster shots within the municipal government and private industry and to set up clinics for the public.

Widespread use of vaccine has been responsible for significant reductions in the incidence rates of several communicable diseases. It is essential not to relax now.

Gwynneth Wilson

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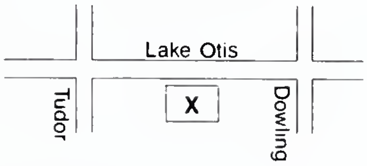

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CERTIFIED MEDICAL ASSISTANTS CAN HELP A PHYSICIAN'S OFFICE PROSPER

The person once called "Gal Friday" in the medical office has become a professional, multi-skilled person and a valued asset to the medical office team. Her title reflects that professionalism: medical assistant.

Once trained on the job, many medical assistants are now being trained in 165 post-secondary programs, usually on college campuses. The programs, which are accredited by the AMA's Committee on Allied Health Education and Accreditation (CAHEA) provide anywhere from six months to two years of training and produce the only allied health professionals specifically trained to work in a physician's office.

Medical assistants perform a wide variety of administrative and clinical duties. In many instances, they actually manage medical emergency situations and medical facilities and/or personnel. Dedicated to assisting in patient care management, they have learned to communicate effectively in providing instruction to patients.

The American Association of Medical Assistants (AAMA)—the oldest and largest medical assistants organization—exists solely to support the improvement of medical assistants through education. With more than 500 chapters in almost every state including Alaska, the AAMA is working to help medical assistants keep their skills up to date in the rapidly changing healthcare field.

The Certified Medical Assistant (CMA) program developed by the organization in 1963, is ensuring that medical assistants prove their professional competence. To date, more than 40,000 medical assistants have passed the strenous examination, resulting in a CMA credential. In addition, CMAs re-validate their certificates every five years, either by accumulating recognized continuing education credits (CEUs) or by a follow-up exam to prove their competence. The National Board of Medical Examiners serves as the AAMA's test consultant.

It's important to recognize that the AAMA was *not* formed for the purpose of members comparing salaries or to eventually become a union. In fact, it is written in the organization's by-laws that the AAMA will *never* become a union. The objective is and always has been to support the improvement of medical assistants through continuing education programs, workshops, seminars and monthly meetings. These programs may include topics such as: Marketing Your Medical Office; Computer Literacy; DRGs and Current Issues; Time Management; Personnel Management; the Aging Process and Risk Management.

Physicians not only serve as advisors to most AAMA chapters, but most meetings also include at least one physician as guest speaker. Many physicians even support membership in the AAMA by underwriting their medical assistant's participation.

In 1983, the AAMA completed its first long-range strategic plan. The plan focuses on the AAMA's need to promote certification for all medical assistants. To this end, the AAMA is developing publications that will help prepare the medical assistants for the certification test. The test, given twice annually at approximately 75 locations around the U.S., is administered by the AAMA. The plan also calls for re-certification every five years for the medical assistant to keep his or her credential (beginning in 1988).

Ultimately, the AAMA hopes that state legislatures will recognize AAMA certification rather than state licensure as a requisite to practice. Given the rapid changes in the healthcare market, state legislatures are likely to address the credentialing of medical assistants in the near future.

The plan also calls for the development of workshops and materials that will assist the medical assistant in professional advancement and leadership skills. A new home study course on law and the medical office was recently introduced, and a course on medical office management will be published shortly. Membership recruitment workshops also are being held.

The real value of well-trained medical assistants is their marketability. Physicians are becoming aware that, from a marketing and even a liability standpoint, medical assistants can make or break the practice. The medical assistant usually is the first and last person to see the patient. Further, the patient often spends more time with the medical assistant than even the physician. Fortunately, many physicians are recognizing the fact that hiring untrained, poorly-qualified personnel can actually harm their practice.

To the contrary, physicians who hire CMAs are finding that their patients **do** receive better care and that their offices **are** managed more effectively. Ultimately, these CMAs may even replace a cadre of other allied healthcare personnel. Their broad skills, training and expertise clearly make them excellent candidates.

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Thiazides may add to or potentiate the action of other antihypertensive drugs.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

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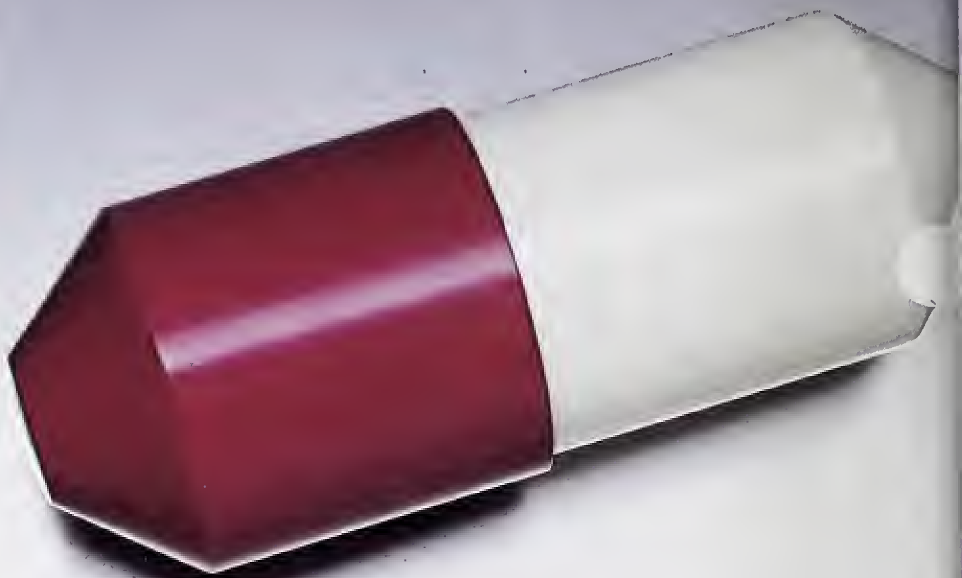

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PRESIDENT'S PAGE

Efforts at Tort Reform in Alaska are beginning to show some effect. Most legislators are now aware of the problem. There is anything but unanimity concerning the solution. In addition to the now familiar proposals for tort reform, there are proposals for no-fault insurance, joint underwriting associations, State reinsurance, patient compensation funds, and broader regulations of insurance companies. A certain vocal segment insists that problems are mostly related to incompetence, ignorance, insensitivity and greed on the part of tortfeasors.

The medical profession in Alaska and other states has significant experience with alternative insurance mechanisms. The Medical Insurance Corporation of Alaska (MICA) has suffered recent adversities. Since its inception, MICA has been a responsible and well managed corporation. The Board of Directors, staff, and volunteer physicians working with MICA are to be commended for their efforts regarding risk management. Nevertheless, risk management in and of itself, is apparently an insufficient solution to the problems of rising insurance premiums, increasingly defensive medicine, and increasing frequency and severity of claims for personal injury. Tort reform is needed.

In our enthusiasm for tort reform, we should not overlook the fact that there are genuine instances of malpractice and physician incompetence. There are patients who are worse off by virtue of their encounter with physicians. The root causes of such instances are many and varied. Instances of insufficient training, inattention to details, substance abuse, psychiatric disorders, personality disorders and gross incompetence have all been documented causes of malpractice cases. Many patients allege that doctors cover for one another in such instances. They argue that such cases are either ignored or whitewashed. It is reasonable to consider whether such allegations are true, and if so, what can or should be done.

Our expert advisory panels have worked well for the most part. Decisions which are adverse to a colleague are painful to render. Nevertheless, most panels have accomplished their duties with integrity. There are exceptions. Some panels have been untimely at best. There are few, if any, excuses for such actions. In order to maintain credibility, panel members must perform their duties in a timely fashion, with a genuine concern for the integrity of the system.

This year the Medical Society has attempted to make our efforts to address the problem of the impared physician more visible. It would appear that the public wants assurance that something is being done to help physicians who are impaired for one reason or another. Your present leadership believes that such efforts should be made more visible to the public-at-large. Your thoughts on this matter are solicited.

There are many proposals around the Nation for increased regulation and stricter licensing requirements of physicians. Many states have adopted mandatory reporting of peer review activities, restriction of staff privileges, adverse outcomes of malpractice actions, and immunity from prosecution to individuals reporting such incidents. If tort reform proposals are successful, there will be, no doubt, a demand for stricter regulation of many activities including medicine. Opinions are strongly held. The answers are anything but certain. We solicit your thoughtful and constructive input as we enter the New Year and the next legislative session.

If asked, we should be ready to respond with prudent and constructive proposals to insure that patients seeking medical assistance are afforded the most reasonable opportunity of a successful outcome. Please direct your thoughts and suggestions to me, the Executive Committee, your councilor, or Martha MacDermaid at ASMA. Thank you for your help and support.

David McGuire, M.D.
President, ASMA

FRIENDS OF MEDICINE

Rarely physicians in the medical community become impaired, be it from alcohol, drugs, emotional illness or senility. ASMA through the Friends of Medicine has established means to help deal with problems of such individuals. If you know of any physician in need of assistance please call the ASMA office at 562-2662. Help rendered early in the course of many illnesses may prevent irreversible damage.

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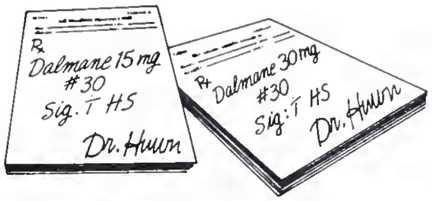

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LOCAL ANESTHESIA AND ARTHROSCOPIC SURGERY OF THE KNEE

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Abstract

We present a technique of local anesthesia for use with arthroscopic surgery of the knee. The results of patients receiving local anesthesia were evaluated by reviewing 751 arthroscopic procedures in 629 patients. This local anesthetic technique was 92% successful and has proved to be an excellent alternative for most if not all types of arthroscopic surgical procedures on the knee.

Introduction

The diagnostic and therapeutic results of arthroscopy have been well documented over the last decade. Until recently, general and spinal anesthesia have been preferred for arthroscopic procedures while local anesthesia has been used less frequently (1,2,3,4,5). Local infiltration anesthesia and intravenous regional anesthesia techniques have been used in arthrotomies and peripheral extremity surgery for quiet some time (3,6,7,8). However, local anesthesia techniques have only lately become more popular in arthroscopic procedures. In 1976 Jackson and Dandy recommended general anesthesia for arthroscopy, but said that in circumstances where local anesthesia was indicated, infiltration of the skin or blocking of the infrapatellar branch of the saphenous nerve with one percent lidocaine, followed by injection of fifty milliliters of saline and ten milliliters of two percent lidocaine intra-articularly was sufficient. (1) Two years later McGinty and Matzer described a technique in which ten milliliters of 0.5 percent lidocaine was injected into the skin and subcutaneous tissues at the point where the arthroscope was to be inserted. Following insertion of the scope, ten milliliters of one percent lidocaine and ten milliliters of 0.5 percent bupivacaine were instilled into the joint space. The authors concluded that "the use of local anesthesia does not, in any way, reduce the accuracy or complications of the procedure" (9). Halperin and coworkers utilized the same technique described by McGinty with a controlled pressure-irrigation system. They concluded that the average time of the examination was reduced substantially by the use of local anesthesia (10). In 1982, Wredmark and Lundh described a local anesthesia technique that utilized a controlled pressure-irrigation with a 0.2 percent prilocaine solution. This was used for arthroscopies in 174 patients, and eighteen of those patients also underwent transcutaneous partial meniscectomies at the same time (11). In 1983, Read published a technique which he used for both diagnostic and operative arthroscopy. The technique involved an intra-articular injection of twenty milliliters of bupivacaine, and the use of a mixture of bupivacaine and lignocaine to infiltrate the skin at the sites of the portals and to block the femoral nerves (12).

In this paper we present a local anesthesia technique used for outpatient arthroscopic examinations and operative arthroscopy. In order for this technique to be a functional alternative to general anesthesia, the surgeon must be quite experienced with the technique of arthroscopy and comfortable with the idea of having the patient awake and talkative throughout the surgery.

Contraindications against the use of local anesthesia for arthroscopic surgery include any known hypersensitivity to the local anesthetic agents and cases in which an open procedure is planned to follow the arthroscopy. A relative contraindication is hesitation on the part of the patient to have the procedure done under local anesthesia. In addition, it has been our experience that children under sixteen to seventeen years of age fare better when they have their surgery under general anesthesia.

Methods

The charts of patients who underwent arthroscopic surgery performed by two of us (D.A.M.) and J.D.F.), between January 1, 1983 and July 25, 1984, were reviewed. The group was composed of 629 patients on whom 751 arthroscopies had been done. This number included 341 right knee operations, 339 left knee operations, 54 bilateral operations, ten repeated right knee operations and seven repeated left knee operations. The patient group was sixty-one percent males and thirty-nine percent females, with a mean age of thirty-three years and an age range of seven to eighty-one years.

Forty-five percent of the operations were done under local anesthesia and fifty-five percent under general. All of the local anesthesia cases were performed with an anesthesiologist standing by.

Table I
Other Medications Used

Drug	No. of Patients	Mean Dose	Standard Deviation	Dose Range
Thiopental Sodium	137	256 mg.	112 mg.	o mg. to 650 mg
Diazepam	67	8.3 mg.	3.6 mg.	1.0 mg. to 20.0 mg.
Fentanyl	168	0.12 mg.	0.06 mg.	0.025 mg. to 0.03 mg.
Droperidol	6	0.92 mg.	0.03 mg.	0.5 mg. to 1.25 mg.
Innovar*	16	0.8 ml.	0.4 ml.	0.5 ml. to 2.0 ml.

*Innovar is a mixture, in a 1:50 ratio, of fentanyl and droperidol. Each milliliter contains 0.05 mg fentanyl and 2.5 mg droperidol. Janssen Pharmaceutical Incorporated.

Prior to surgery, a history was taken and a physical examination was done on each patient. All of the surgeries were performed either at a free-standing outpatient surgical facility, or at one of the two hospitals in Anchorage, Alaska.

When a patient arrived at the surgical facility, his knee was shaved and a concentrated povidone-iodine gel (Solo-prep, Parke-Davis and Company) applied. The patient was then taken to the operating room, where the anesthesiologist administered sedation, usually in the form of a small dose of thiopental sodium. Table 1 shows the mean dose and dose range of the various sedative drugs and pain control medications that were used with this group of patients. During the first half of this study period, other medications were used routinely for sedation, including diazepam and a combination of diazepam and fentanyl. About halfway through the study period, we began using thiopental sodium alone, which induces a short lived sedation during which the intra-articular and the subcutaneous injections are given. Most patients awaken completely in about fifteen minutes, and usually have no memory of the injections.

While the patient was sedated, the surgeon made a second application of Solo-prep and then injected the local anesthetic. Thirty milliliters of 0.5 percent bupivacaine with einephrine (Marcain, Sterling Drug Incorporated) were injected intra-articularly with an eighteen gauge needle, at a point just proximal and lateral to the patella. The ease of flow of the bupivacaine is an indication of proper placement of the needle. Twenty milliliters of one percent lidocaine with epinephrine (Xylocaine, Astra Pharmaceutical Products Incorporated) was used to infiltrate the skin and subcutaneous tissues with a twenty-two gauge needle where each of the three portals would be made. The twenty milliliters is divided approximately equally among the three sites. The injection for the proximallateral portal was placed approximately one to two centimeters proximal to the site of the intra-articular injection. The injection for the lateral portal was positioned in the soft tissue, near the distal pole of the patella, at the apex of a triangle bounded by the lateral margin of the lateral femoral condyle, the lateral margin of the patella, and the margin of the lateral tibial plateau. (See photo) The injection for the antero-medial portal was made sufficiently proximal to the tibial plateau to allow for clearance of the operating instruments over the meniscus once the portal was established. There was some variation from patient to patient in the amount of lidocaine used to anesthetize the portals. The mean total dose in this patient sample was 19.7 milliliters and the standard deviation was 4.2 milliliters. The dose range was ten to forty milliliters. Two of the fifty-four bilateral procedures were done under local anesthesia. The first of these two patients received fifteen milliliters of 0.5 percent bupivacaine and fifteen milliliters of one percent lidocaine in each knee. The second patient received twenty milliliters of 0.5 percent bupivacaine and ten milliliters of one percent lidocaine in the right knee and twenty milliliters of bupivacaine and eighteen milliliters of lidocaine in the left. We encountered no instances of anesthetic toxicity.

After the anesthetic was administered, a pneumatic tourniquet was placed around the patient's thigh but was left deflated. The patient's thigh with the tourniquet in place was put into an Anspach surgical leg holder (Depuy, Warsaw, Indiana) and his knee was passively flexed several times to circulate the bupivacaine throughout the joint space.

The surgery was then carried out with the arthroscope placed in the lateral portal, the outflow cannula in th e proximal-lateral portal and the operating instruments in the anteromedial portal. Positive pressure-irrigation was achieved using an infusion pump (Gambro model Ak-10, Lune, Sweden) with the inflow through the arthroscope.

Results

Twenty-seven of the 335 local anesthesia cases (eight percent) required subsequent general anesthesia at some point during the procedure, resulting in an overall success rate of ninety-two percent for the local anesthesia technique.

All 751 procedures included a complete arthroscopic examination. Sixty-seven of those involved no other procedures except the diagnostic arthroscopy, and 684 involved additional arthroscopic surgical procedures. The highest success was found with excision of synovial plicas (98 percent) and the lowest with removal of loose bodies (78 percent). Loose body removals were found to have significantly lower local anesthesia success than any other operation (Table 2). The reasons for this will be discussed later.

Table II
Local Anesthesia Success Rates
For Operative Arthroscopy

Procedure	Success Rate
Diagnostic arthroscopy	88%
Partial lateral meniscectomy	87%
Partial medial meniscectomy	92%
Debridement of chondral surfaces	96%
Removal of loose body(s)	78%
Lateral retinacular release	87%
Excision of synovial plica	98%
Osteophytectyomy	89%
Partial synovectomy	90%

Fourteen patients had mild complications secondary to the local anesthetic, for an overall complication rate of 4.2 percent. Eleven patients (3.3 percent) developed a superficial erythema in a pattern typical of the distribution of the anesthetic. This rash has the appearance of a mild sunburn and occasionally proceeds to a stage of superficial peeling of the skin similar to that seen in sunburn. One patient developed some bruising due to the epinephrine effect. One patient had an allergic reaction to the lidocaine which presented as a topical dermatitis to the typical pattern of distribution of the lidocaine over the three portals. One patient developed some blanching of the skin in the area of distribution of the epinephrine.

Two postoperative infections developed in the sample of 751 arthroscopies. One of these patients had his surgery under general anesthesia and the other was done under local.

Discussion

A safe, dependable local anesthesia technique is valuable to both the surgeon and the patient for several reasons. It allows the patient to be awake throughout the procedure and to observe on the monitor exactly what is wrong with his knee, and what the surgeon is doing to correct his problem. In addition, there is no lengthy recovery from general anesthesia, and the patient can usually leave the surgical facility within an hour postoperatively. The use of a local anesthetic is valuable to the surgeon because it increases patient participation which leads to a greater patient acceptance of the surgical procedure. This results in an easier course of rehabilitation for the patient.

The use of an epinephrine containing anesthetic also eliminates the need for a tourniquet, thereby decreasing any complications which may be tourniquet related. Other potential complications that are avoided by the use of a local anesthetic include the risk of injuring ligaments when placing varus and valgus stress on the leg to open the joint space.

This risk is presumably decreased because the patient is awake and can feel excessive stress on the ligaments.

The use of local anesthesia is not without potential problems, however. For example, inadequate visualization due to muscle tension, causing inadequate opening of the joint, could possibly result in increased scoring of the articular surfaces with the surgical instruments. Additionally, anxiety on the part of both the patient and surgeon may adversely affect the outcome.

The presence of an anesthesiologist in the operating room throughout the surgery helps to provide control over the operation in several ways. In those instances where matters are not progressing well for whatever reason, we do not hesitate to resort to general anesthesia. Should general anesthesia be needed at any time during the surgery, or if additional sedation is needed to help and anxious patient, the anesthesiologist is there. In addidtion, the anesthesiologist is very valuable for talking the patient through the procedure. Anesthesiologists are employed in our setting, but in those instances where nurse anesthetists are routienly used would undoubtedly work well.

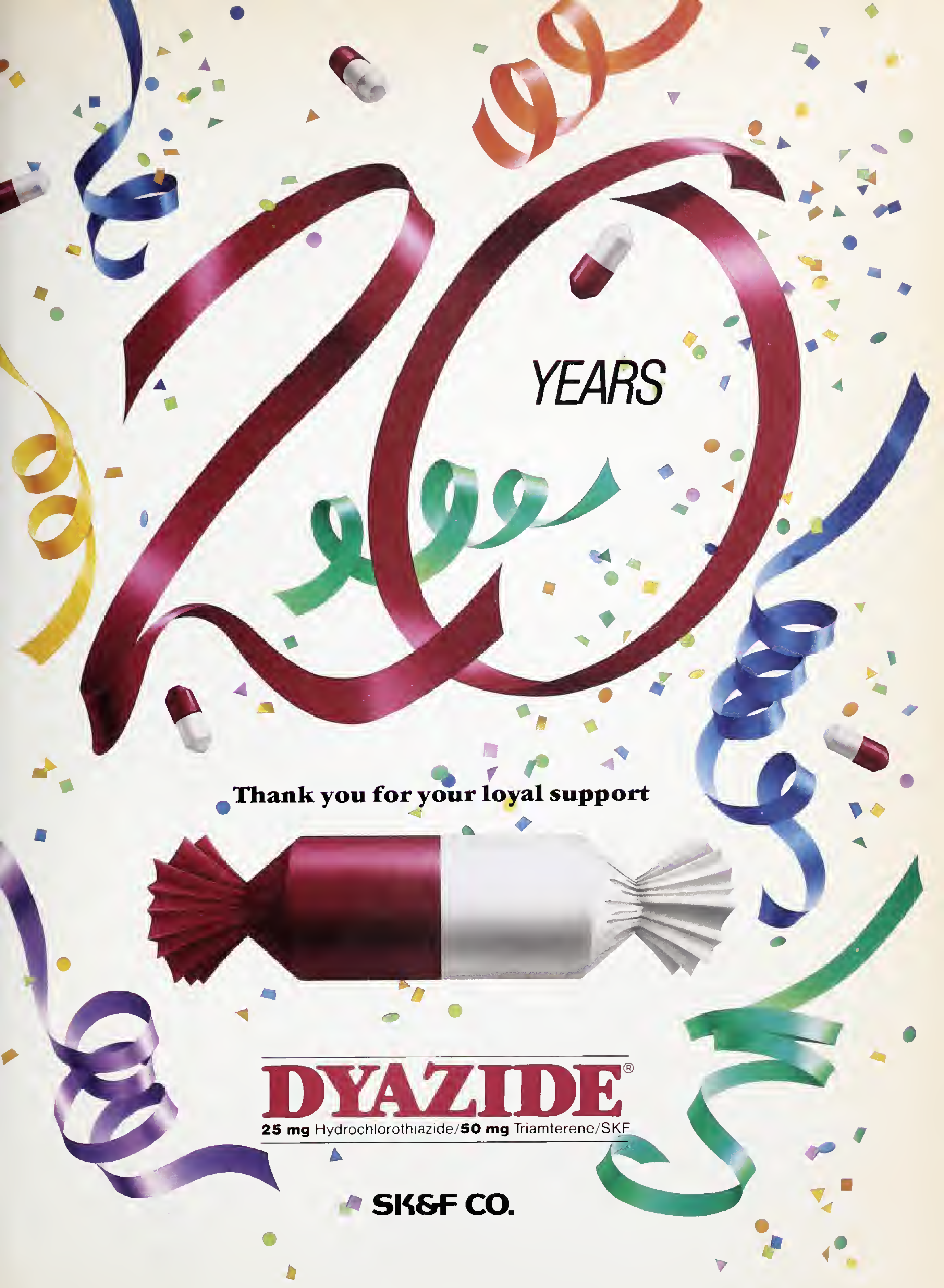
The use of small doses of thiopental sodium for sedation while the intra-articular injection is given has become a part of our technique. However, the use of thiopental is not a requisite of the local anesthesia procedure and this technique can certainly be done well without it.

The basic technique involves both an intra-articular injection of a long acting local anesthetic and local infiltration of the skin over the portal sites with a shorter acting drug. There has been criticism from some anesthesiologists that the total milligram dose of the local anesthetic agents we have been using exceeds the safe dose for patients. Smaller doses of anesthetic agents may work as well as those employed. In the absence of complications, we have had no reasons to employ different doses. We believe the reason the problem is avoided is that probably little of the bupivacaine is absorbed into the general circulation from the intra-articular space. Secondly, the lidocaine contains epinephrine, so its absorption is significantly slowed as well.

There was only one case of significant bruising due to the effects of the epinephrine in the local anesthetic observed in this series of patients. Additionally, there were eleven cases of superficial erythema over the area of anesthetic distribution. In cases performed subsequent to this series, we have observed both bruising and erythema, but have never seen these go on to cause other problems. We are aware of reports of full thickness skin necrosis in conjunction with the use of local anesthesia. In our experience, we have encountered no instances of skin necrosis, wound dehiscence or infection which we can attribute to the use of local anesthesia. In very few instances do we use more than seven milliliters of one percent lidocaine injected in the area of an intended portal. This causes a wheal to be raised of approximately 1.0 to 1.5 centimeters in diameter.

In this series of patients, our success rate with local anesthesia for removing loose bodies was significantly lower than for other procedures. This is explained in part by the fact that loose body removals often require the establishment of a postermedial or posterolateral portal during the surgery. Our technique has improved since this study was done, and it has become easier to make posteromedial and posterolateral portals by secondary injection during the surgery. We have observed no adverse reactions to the use of additional anesthetic for this purpose. However, posterolateral portals should only be established using lidocaine rather than the longer acting bupivacaine. We have encountered one instance of transient total peroneal nerve palsy which lasted four hours without any residual sequelae.

Recently, we have been increasingly successful in doing arthroscopic lateral retinacular releases under local anesthesia. The local anesthesia success rate for lateral releases in this patient series was eighty-seven percent. The greatest success of this procedure comes when the area of intended release is injected with 0.5 percent bupivacaine at the same time the intra-articular injection is given. Ten milliliters of 0.5 percent bupivacaine is injected along the line of intended release and the remaining twenty milliliters is injected intra articularly. Then the portals are injected with the normal dose of one percent lidocaine. Thus the total dose of the two local anesthetic agents are no different from any other procedure. An added benefit of injecting the bupivacaine along the line of release at the start of the surgery is that this allows plenty of time for the the drug to reach its maximum anesthetic ef-



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WyTensin®
(guanabenz acetate)

Antihypertensive therapy that does not increase cholesterol

See important information on following page.

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that does not increase cholesterol

Brief Summary

Before prescribing, consult the complete package circular.

Indications and Usage: Treatment of hypertension, alone or in combination with a thiazide diuretic

Contraindication: Known sensitivity to the drug.

Precautions: 1 Sedation. Causes sedation or drowsiness in a large fraction of patients When used with centrally active depressants, e g , phenothiazines, barbiturates and benzodiazepines, consider potential for additive sedative effects 2 Patients with vascular insufficiency: Like other antihypertensives use with caution in severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease, or severe hepatic or renal failure 3. Rebound Sudden cessation of therapy with central alpha agonists like **Wytensin** may rarely result in "overshoot" hypertension and more commonly produces increase in serum catecholamines and subjective symptomatology

INFORMATION FOR PATIENTS Advise patients on **Wytensin** to exercise caution when operating dangerous machinery or motor vehicles until it is determined they do not become drowsy or dizzy Warn patients that tolerance for alcohol and other CNS depressants may be diminished Advise patients not to discontinue therapy abruptly

LAB TESTS In clinical trials, no clinically significant lab test abnormalities were identified during acute or chronic therapy Tests included CBC, urinalysis, electrolytes, SGOT, bilirubin, alkaline phosphatase, uric acid, BUN, creatinine, glucose, calcium, phosphorus, total protein, and Coombs' test During long-term use there was small decrease in serum cholesterol and total triglycerides without change in high-density lipoprotein fraction. In rare instances occasional nonprogressive increase in liver enzymes was observed, but no clinical evidence of hepatic disease

DRUG INTERACTIONS. **Wytensin** was not demonstrated to cause drug interactions when given with other drugs, e g , digitalis, diuretics, analgesics, anxiolytics, and antiinflammatory or antiinfective agents, in clinical trials However, potential for increased sedation when given concomitantly with CNS depressants should be noted

DRUG/LAB TEST INTERACTIONS No lab test abnormalities were identified with **Wytensin** use

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY No evidence of carcinogenic potential emerged in rats during a two-year oral study with **Wytensin** at up to 9 5 mg/kg/day, i e., about 10 times maximum recommended human dose In the Salmonella microsome mutagenicity (Ames) test system, **Wytensin** at 200-500 mcg per plate or at 30-50 mcg/ml in suspension gave dose-related increases in number of mutants in one (TA 1537) of five *Salmonella typhimurium* strains with or without inclusion of rat liver microsomes No mutagenic activity was seen at doses up to those which inhibit growth in the eukaryotic microorganism, *Schizosaccharomyces pombe*, or in Chinese hamster ovary cells at doses up to those lethal to the cells in culture In another eukaryotic system, *Saccharomyces cerevisiae*, **Wytensin** produced no activity in an assay measuring induction of repairable DNA damage Reproductive studies showed a decreased pregnancy rate in rats given high oral doses (9 6 mg/kg), suggesting impairment of fertility Fertility of treated males (9 6 mg/kg) may also have been affected, as suggested by decreased pregnancy rate of mates, even though females received drug only during last third of pregnancy

PREGNANCY Pregnancy Category C WYTENSIN® MAY HAVE ADVERSE EFFECTS ON FETUS WHEN ADMINISTERED TO PREGNANT WOMEN A teratology study in mice indicated possible increase in skeletal abnormalities when **Wytensin** is given orally at doses 3 to 6 times maximum recommended human dose of 1 0 mg/kg These abnormalities, principally costal and vertebral, were not noted in similar studies in rats and rabbits However, increased fetal loss has been observed after oral **Wytensin** given to pregnant rats (14 mg/kg) and rabbits (20 mg/kg) Reproductive studies in rats have shown slightly decreased live-birth indices, decreased fetal survival rate, and decreased pup body weight at oral doses of 6 4 and 9 6 mg/kg There are no adequate, well-controlled studies in pregnant women **Wytensin** should be used during pregnancy only if potential benefit justifies potential risk to fetus.

NURSING MOTHERS Because no information is available on **Wytensin** excretion in human milk, it should not be given to nursing mothers

PEDIATRIC USE Safety and effectiveness in children less than 12 years of age have not been demonstrated, use in this age group cannot be recommended

Adverse Reactions: Incidence of adverse effects was ascertained from controlled clinical studies in U S and is based on data from 859 patients on **Wytensin** for up to 3 years There is some evidence that side effects are dose-related Following table shows incidence of adverse effects in at least 5% of patients in study comparing **Wytensin** to placebo, at starting dose of 8 mg b i d

Adverse Effect	Placebo (%) n = 102	**Wytensin** (%) n = 109
Dry mouth	7	28
Drowsiness or sedation	12	39
Dizziness	7	17
Weakness	7	10
Headache	6	5

In other controlled clinical trials at starting dose of 16 mg/day in 476 patients, incidence of dry mouth was slightly higher (38%) and dizziness was slightly lower (12%), but incidence of most frequent adverse effects was similar to placebo controlled trial. Although these side effects were not serious, they led to discontinuation of treatment about 15% of the time In more recent studies using an initial dose of 8 mg/day in 274 patients, incidence of drowsiness or sedation was lower, about 20% Other adverse effects reported during clinical trials but not clearly distinguishable from placebo effects and occurring with frequency of 3% or less Cardiovascular—chest pain, edema, arrhythmias, palpitations Gastrointestinal—nausea, epigastric pain, diarrhea, vomiting, constipation, abdominal discomfort Central nervous system—anxiety, ataxia, depression, sleep disturbances ENT disorders—nasal congestion Eye disorders—blurring of vision Musculoskeletal—aches in extremities, muscle aches Respiratory—dyspnea Dermatologic—rash, pruritus. Urogenital—urinary frequency, disturbances of sexual function Other—gynecomastia, taste disorders

Drug Abuse and Dependence: No dependence or abuse has been reported

Overdosage: Accidental ingestion caused hypotension, somnolence, lethargy, irritability, miosis, and bradycardia in two children aged one and three years. Gastric lavage and pressor substances, fluids, and oral activated charcoal resulted in complete and uneventful recovery within 12 hours in both Since experience with accidental overdosage is limited, suggested treatment is mainly supportive while drug is being eliminated and until patient is no longer symptomatic Vital signs and fluid balance should be carefully monitored Adequate airway should be maintained and, if indicated, assisted respiration instituted No data are available on **Wytensin** dialyzability

Dosage and Administration: Individualize dosage. A starting dose of 4 mg b i d is recommended, whether used alone or with a thiazide diuretic. Dosage may be increased in increments of 4 to 8 mg/day every one to two weeks, depending on response Maximum dose studied has been 32 mg b i d , but doses this high are rarely needed

How Supplied: (guanabenz acetate) Tablets, 4 mg, bottles of 100 and 500; 8 mg and 16 mg, bottles of 100 Revised 2/14/85

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6. The lines are thirteen blocks long.
7. My mother won't let me.
8. I didn't sign up.
9. I'm going out of town.
10. Asthma runs in my family.
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fect before the release is done.

In our work we utilize an infusion pump to provide positive pressure irrigation. This gives the surgeon a high degree of control over the irrigation of the joint space and aids in hemostasis. Additionally, we have found the pump to be advantageous because we use a four millimeter arthroscope. Whatever the preferred method of irrigation, if the surgeon finds it satisfactory when used with general anesthesia, it will be satisfactory when used with local anesthesia. What is important is that the irrigation provide adequate distension and clearance of the joint space to allow for satisfactory visualization.

The use of a leg holder with a deflated tourniquet during arthroscopies is the perference of the authors but any other type of leg holder will aslo work. Contrary to some reports we have not been troubled by increased bleeding when working without an inflated tourniquet.

We have presented a local anesthesia technique that we have been using for diagnostic and operative arthroscopy with a high degree of success. We have been doing many types of procedures under local anesthesia quite routinely without any complaints of pain or discomfort. Examples include debridement of joint surfaces, excision of synovial plicas, partial medical meniscectomies and partial synovectomies.

In those cases where local anesthesia failed, the most common reason for failure was patient restlessness and anxiety rather than pain. Of utmost importance to the success of this technique is an understanding, cooperative and well informed surgical team.

Conclusion

Local anesthesia can be a successful alternative to general or regional anethesia in diagnostic and operative arthroscopy of the knee. We have described a technique for local anesthesia which has proven 92 percent successful in a large (629) patient sample.

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INFANT APNEA SYNDROMES PART III

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> *"It is, however, a well verified fact that a cat will kill an infant by stealing its breath...By some it is supposed that the attraction is the milkladen breath of the child; but the regularity with which it steals the pure air and denies a fresh supply, indicates a more vicious and wicked purpose."*
>
> *Shaftesbury 1898*

Introduction

Our previous articles (5,6) in the Journal have discussed terminology related to infant apnea syndromes and the Sudden Infant Death Syndrome (SIDS), the etiologies of recurrent apnea, and the pathophysiology of chronic hypoxia. This paper will demonstrate polygraphic patterns of normal and abnormal ventilatory control, present SIDS mortality and recurrent apnea incidence statistics, explain the purpose and services of the Apnea Monitoring Program, present interviews with parents who have used a home apnea monitor, and offer a program for follow-up care of this high risk group of infants.

Apnea is the cessation of air exchange due to either an arrest of respiratory activity or the lack or airflow. Characteristically, this has been described as central or obstructive in nature. The importance of such events is dependent on gestational age and the accompanying cardiovascular changes.

Significant apnea is defined as cessation of airflow for greater than nineteen seconds or a shorter duration if associated with bradycardia and / or cyanosis (table I).

The frequency of apnea is also important in assessing a pathologic state, since infants between 1 and 6 months have been found to display frequent short apneic events of three to ten seconds (2).

Table I
Infantile Apnea

Author	Definition of Apnea
Steinschneider '72	► 15 seconds
Guilleminault & Anders '76	► 10 seconds
Shannon '77	► 20 seconds
Steinschneider '77	► 20 seconds
Cornwell '78	► 10 seconds
Hoppenbowers '78	► 15 seconds
Brady '78	near miss event
Kelly & Shannon '79	near miss
Guilleminault & Korobkin '79	near miss
Peabody '79	significant decrease in Tcp02
Haddad '81	near miss
Southall '82	► 15 seconds
American Academy of Pediatrics	► 20 seconds
Kelly & Shannon '79	► 15 seconds and cardiovascular changes

Several prevailing theories explaining the pathophysiology of infant apnea include: A) hypoventilation seen in some infants may lead to hypoxemia and apnea which is mediated by the central nervous system (4); B) apnea may be related to abnormal thiamine metabolism affecting neurochemistry (10); C) vagal stimulation to the upper third of the esophagus during gastroesophageal reflux (1,9); D) neck flexion causing upper airway obstruction (13); and E) overproduction or activity of endorphins causing respiratory depression in susceptible infants (8).

Etiologies

Any infant who presents with an apneic event, whether observed by non-medical or medical personnel, should have a minimal evaluation to determine the etiology. An infant who has had one such event is at a twenty to fifty percent risk of having another event. Most of the initial work-up may be accomplished as an outpatient; however, an infant who presents immediately after an apneic episode, a "near miss for SIDS event" (a lifethreatening event), should be presumed to have had a prolonged apneic event which was lifethreatening. This infant should be hospitalized. During this hospitalization, which may be in the community hospital, routine laboratory evaluation should include tests in table II. Caution should be used when interpreting the blood count since an elevation and left shift may occur when an infant has gone through a significant stress such as an apneic event. Therefore, a leukocytosis with a left shift may not indicate infection. An arteriolized capillary blood gas sample may indicate the degree of ischemia and hypoxia following an apneic event. Often, a capillary pH below 7.30 indicates a significant ischemic episode. A normal capillary blood gas does not exclude an apneic event. A lumbar puncture should be done if there are sufficient signs of systemic infection or abnormal neurologic activity. Serum sodium, potassium, calcium and urea nitrogen are recommended to screen for renal dysfunction. Since infantile apnea may be a sign of a seizure disorder, and electroencephalogram should be completed (14). The misinterpretation of the EEG can be a difficult problem to avoid since normal variations are common. A normal EEG does not exclude a seizure disorder in infants at this age. One such infant was referred to our program for evaluation. He had two normal electroencephalograms before a third showed a classic epileptogenic foci. The Wolfe-Parkinson White Syndrome, extra-systoles and congenital heart disease may result in bradycardia, ischemia to the central nervous system and respiratory compromise. A thirteen lead electrocardiogram may be helpful in distinguishing these etiologies. Again, a cardiologist skilled in reading pediatric ECG's is necessary. A roentograph of the chest may help determine infection, aspiration or cardiomegaly. When no definable etiology for the apneic event is clear, the practitioner will be faced with pursuing a more exaustive evaluation. A barium swallow or nuclear radioisotope scan may characterize gastroesophageal reflux, but our method of choice is continuous intra-esophageal pH trending. This method provides quantification of the frequency, duration and respiratory association of reflux and may be used to re-examine pre and post operative acid reflux. We generally do not recommend any gastroesophageal surgery without first completing a pH trending and intraesophageal manometry.

Table II

Lab Evaluation

- CBC with Differential
- Serum Electrolytes
- BUN
- Electrocardiogram
- Electroencephalogram
- Chest Radiograph
- Capillary Blood Gas
- Lumbar Puncture
- Sepsis Workup

If we support central or obstructive apnea as the etiology, a simultaneous polygraphic trending of the infant's cardiorespiratory status, transcutaneous oxygen saturation and behavioral activity are obtained. The infant is admitted to a hospital setting for these procedures. Direct observation of some of the infant's sleep is needed to make a valid determination in distinguishing obstructive from central apnea.

Our future testing facility will help us quantitate this disturbance with more accuracy. Impedance pneumography (trending of heart rate and respirations alone) cannot adequately distinguish central from obstructive apnea. During an obstructive event, an infant will attempt to breathe against the obstructed airway. This will produce changes in impedance and record as respiratory effort. For this reason, it is sometimes necessary to recommend further testing in the sleep laboratory at the Children's Orthopedic Hospital in Seattle, Washington. They have a complete infant sleep center capable of making difficult diagnostic decisions and appropriate therapeutic interventions.

Respiratory Physiology

In our attempt to understand abnormalities of breathing regulation in this high risk group of infants, a few words must be mentioned about normal respiratory physiology. Fetal respiratory movement is dependent on gestational age and becomes more organized during the third trimester. Normally respirations lack regularity and are not dependent on maternal or fetal blood gas tensions. There is periodicity and dependence on fetal rapid eye movements during sleep (figure 1,2). The lack of periodicity or response to stress is an indication of fetal compromise. Some investigators believe that infants with abnormal respiratory controls (those with recurrent apnea) have had disturbances in their respiratory controls in utero. This theory has not been adequately determined.

The normal infant breathing pattern varies with the state of activity. Recordings of respiratory and heart rate (cardiorespirogram) for various states of activity and sleep are depicted in figures 3 and 4.

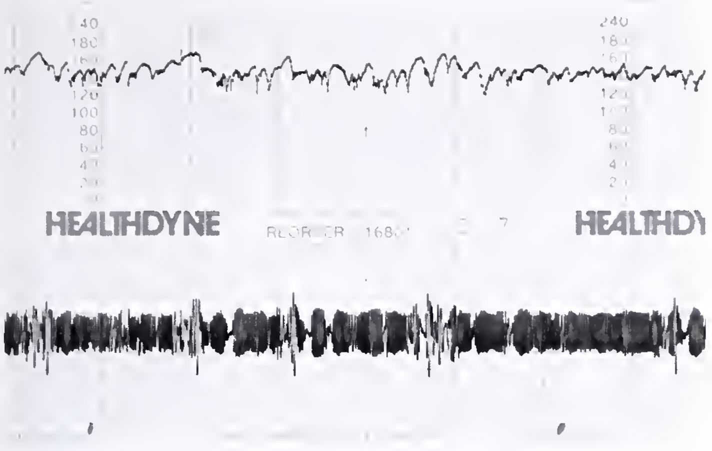


Figure 1 REM Sleep, Stage Unknown

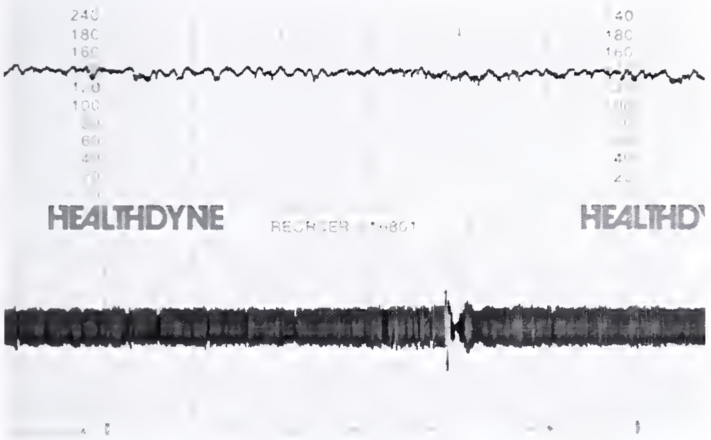


Figure 2 NREM Sleep

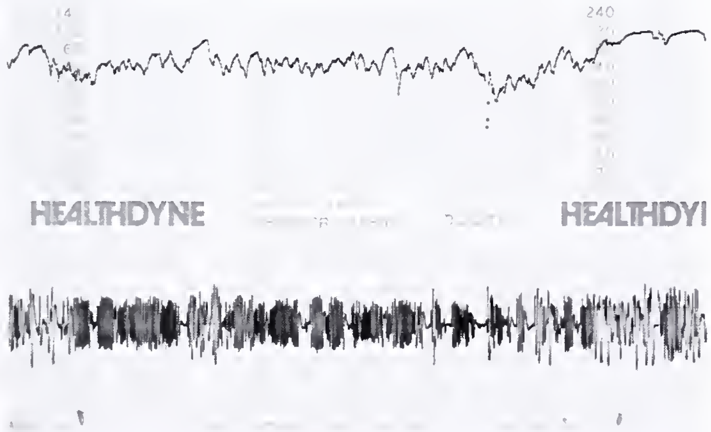
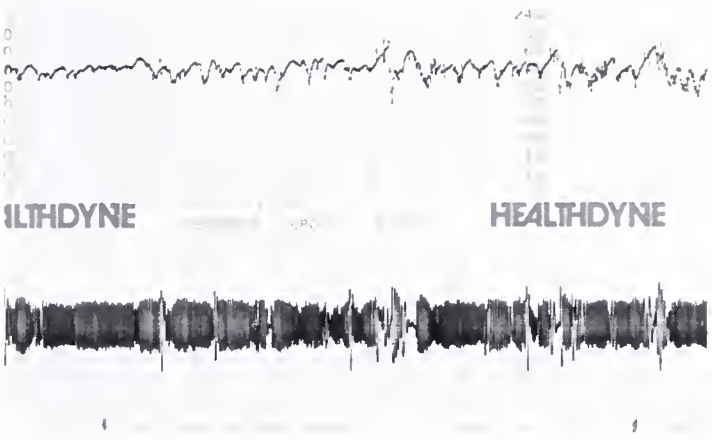


Figure 3 Active Awake



Fugure 4 Quiet Awake

The most frequently observed regular, irregularity in infant respirations is periodic breathing. We have had several referrals after an anxious parent observed, "my baby seems to stop breathing quite often, but starts back on his/her own after awhile" (figure 5). Abnormal patterns of respiration may be trended over 12 to 24 hours. A continuous recording will determine the frequency and duration of apnea as well as associated heart rate changes. Typical abnormal patterns are shown in figures 6, 7 and 8.

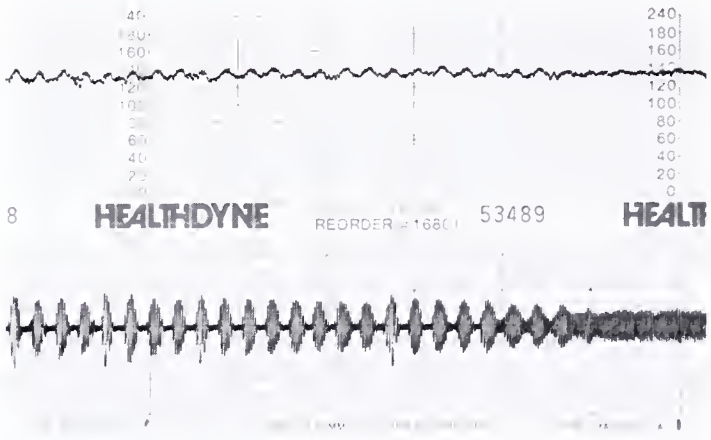


Figure 5 Periodic Breathing

Alaska Mortality

Prior to 1974 Sudden Infant Death Syndrome was not identified as a cause of infant mortality in Alaska. In 1976 the incidence of SIDS in the United States was reported as 2.5/1000 live births (3). That same year the incidence among Alaska natives was reported as 2.3/1000 live births (3). In 1977 and 1980 the Alaska SIDS incidence rates are estimates because there was inconsistent reporting. Table III is the bar graph comparison of SIDS deaths to total infant deaths in the state. Since 1980 the proportion of SIDS appears to be increasing. The reason for this is not known.

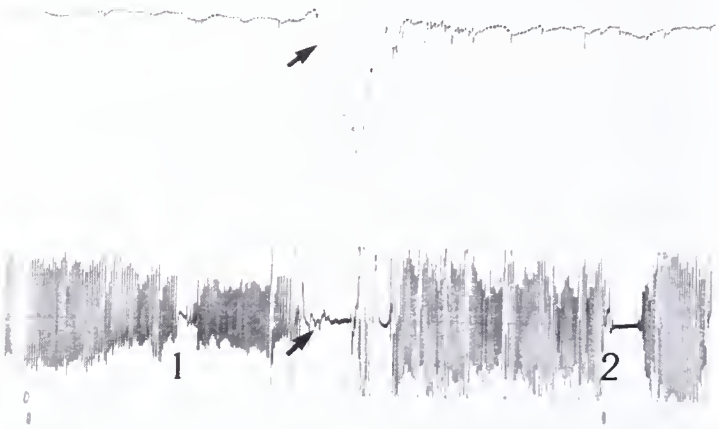


Figure 6

A 30 second apnea and simultaneous bradycardia are seen at arrows. Also, note the short apneic episodes at 1 and 2.

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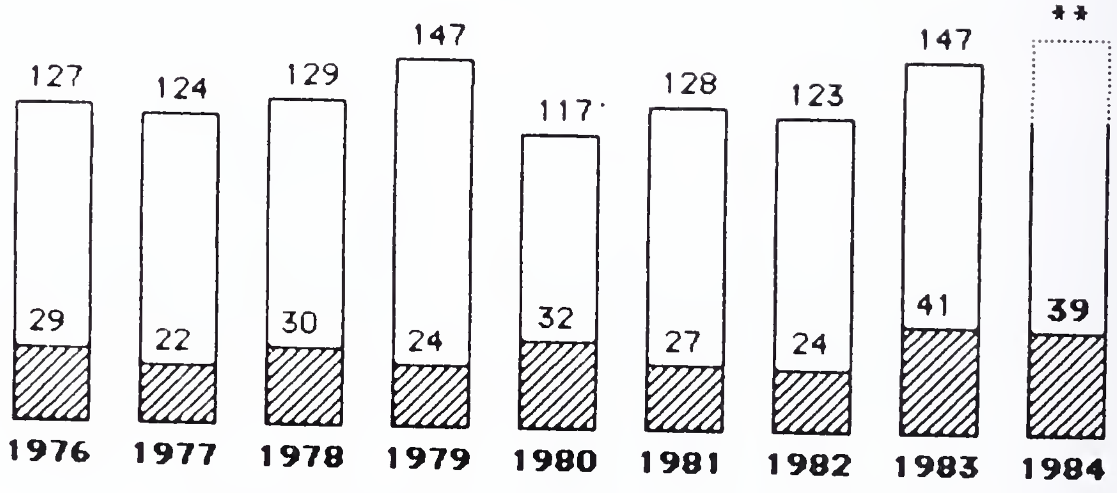
Table III



Infant Deaths per year in Alaska *

Shaded areas are SIDS deaths (reported)

** not available at time of publication *Section of Family Health, Vital Statistics unit

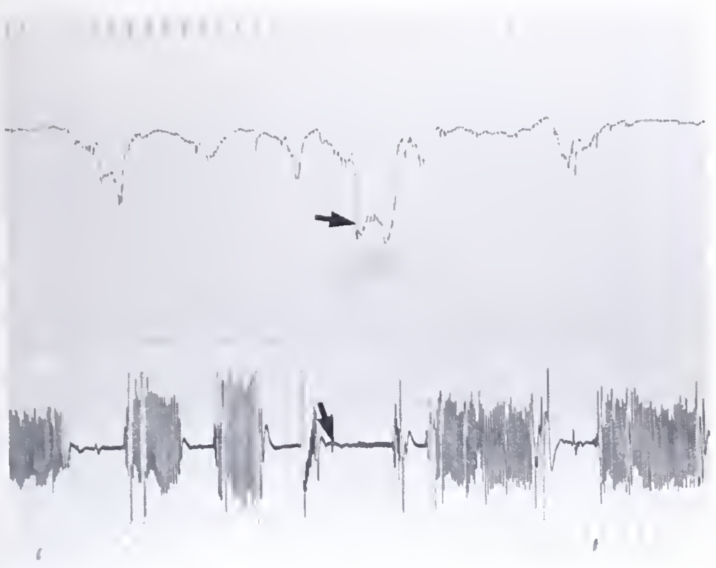


Figure 7
Significant prolonged apnea of 40 seconds duration; insignificant cardiac deceleration to 105 beats per minute.

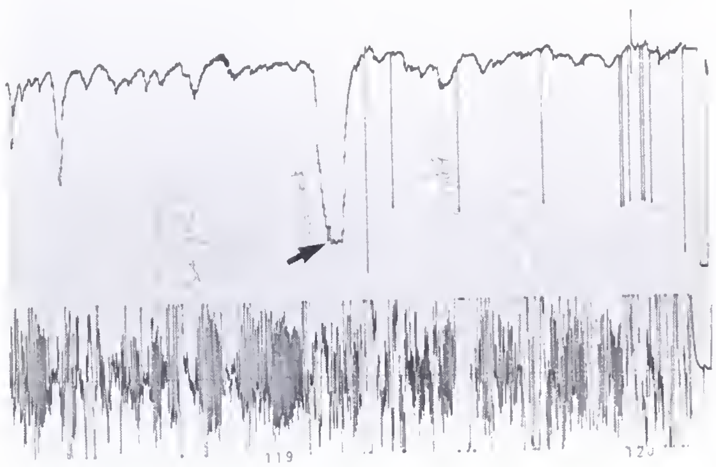


Figure 8
Significant bradycardia to 65 beats per minute of 8 seconds duration, occasionally associated with feedings. Note sawtoothed suckling pattern prior to this.

Apnea Monitoring Program

In 1983 the Apnea Monitoring Program in Anchorage evaluated 59 infants. Forty infants were patients in the Neonatal Intensive Care Nurseries, 19 were state-wide referrals. In 1984 there were 77 infants who had some modality prescribed (Tabel IV). A two channel impedance home monitor was used by 27%; methylxanthine was prescribed for 27%; and forty-six percent were prescribed methylxanthine and home monitoring (Table IV). Table IV demonstrates that the trend has been to prescribe fewer apnea monitors and more methylxanthine therapy. This trend is consistent with the Seattle Children's Orthopedic Hospital experience.

Using random interview techniques (nonscientific) and accumulated patient data, we have concluded that compliance is 36% better when a monitor is prescribed than theophylline alone. Also, 10% of the monitored infants required at least one resuscitation at home.

Most of the infants evaluated by the Apnea Monitoring Program continue to be from the Anchorage area (42% in 1984). For a breakdown of geographic locations see table V.

Since the majority of evaluated infants come from the intensive care nurseries, it is not surprising that the predominant etiology for an apneic event is apnea of prematurity. The distribution of the remaining etiologies (tabel VI) is similar to those previously reported (1).

Etiologies for an apneic event are being better represented in 1985 because of an increase in pediatric referrals, availability of diagnostic services, and early discharge from the intensive care nurseries of infants with oxygen dependent bronchopulmonary dsyplasia. Our recommendation for monitoring this latter group of infants followed two sudden deaths shortly after discharge and an unexpected cardiac arrest of hospitalized patient.

Apnea Evaluation Process

A new patient at either Providence or Humana Hospital will be examined by the director or his colleagues after taking a thorough history. Often, a parental description of the event will determine in which direction a medical evaluation will be pursued. When an etiology cannot be found from the laboratory data, polygraphic testing is done. Two channel impedance, transcutaneous oxygen and / or carbon dioxide monitoring and nursing observations are the mainstay of this process. The family is also supported during this time by the social service department. Families of children who have suffered a life threatening event (LTE) are often in need of emotional support. This may include listening to a description of the event or investigating the families' need for home health care support after discharge from the hospital. Once the diagnosis is agreed upon, the physician determines the need for home monitoring and / or medication in consultation with the program coordinators and the family. The apnea nurse coordinators initiate the teaching necessary and coordinate home needs.

The family training process takes three to four days. It includes a review of the infant's diagnosis, training in the use of the apnea monitor (Healthdyne 16900), infant CPR and respiratory assessment skills. We use situational role playing in teaching how to respond to monitor alarms and when to notify the physician or Emergency Medical Services. Distinguishing real and false alarm states and monitor troubleshooting are also an important part of the training. On the last day of training we requrie the family to "room in" with their baby. During this time they are responsible for their child's care but have medical and nursing backup to answer questions if necessary.

Since parents often need a respite during the monitoring period, we arrange for teaching a relief caretaker if one is available. This is helpful in minimizing possible negative impacts of infant monitoring.

A public health nurse referral is made for the family prior to discharge. The primary physician is also made aware of family support services that were arranged (homemaker, home health care, respite care, etc.), any clarification regarding the monitor or medication, follow-up, and re-evaluation needs for the child. The coordinators are available seven days a week, twenty-four hours a day, for troubleshooting or replacing monitoring equipment.

A recommendation for following the child's cardiorespiratory status is determined prior to discharge. Outside of the Anchorage area, the public health nurses and home health nurses are very helpful in setting up the trend recordings for an out-patient re-evaluation. Utilizing the public health nurses for re-evaluation means a savings of three hospital days per month and allows the family to stay in their community.

Once a normal re-evaluation has been obtained, the infant can be weaned from the monitor. For some parents this is no problem, but for others it is a time of renewed anxiety.

Family Dynamics

Since the advent of home monitoring, there has been a great deal of concern and speculation about its' psychosocial impacts upon the family unit. There have been questions raised as to the effectiveness in preventing death, interference with mother-infant bonding, and marital difficulties. To some these are strong arguments against home monitoring, yet others support it as a motivator in the development of psychosocial support for the high risk infant monitor family (12).

In order to assess some of the impacts upon monitoring families in Alaska, the Apnea Monitoring Program distributed questionnaires to evaluate 1) levels of stress prior to and after the monitoring period, 2) areas which were perceived to be most stressful, 3) comfort in performing CPR in an emergency situation, 4) support systems utilized, 5) financial concerns, and 6) marital difficulties attributed to monitoring.

Most families agreed that the first week of monitoring at home was the most stressful and the time at which the most support was needed. The second most stressful period was felt to be after the monitor was discontinued. Most families relied heavily upon their spouses, relatives, friends, family physician and the apnea nurse coordinators for support.

The families' greatest fears were 1) performance during an emergency, 2) sleeping through an alarm, and 3) a monitor malfunction. Although performance during an emergency was rated as very important, CPR instruction alleviated anxiety in the majority of families.

Most families reported "no change" in their marital relationship and that monitoring "made their life more comfortable with their child". As expected, financial concerns were directly related to third party coverage.

Three families, each with a different reason for monitoring their infant, were interviewed. The families included: 1) a sibling of a SIDS victim, 2) life threatening event (LTE) requiring resuscitation, and 3) apnea of prematurity (AOP).

All families agreed that the first week was the most stressful. They also utilized similar sources of support, while their greatest fear was "performance in an emergency". The first two families also conveyed fears of "monitor malfunctions" and an underlying mistrust of the monitor. The third family of the premature infant did not express this as very concerning and seemed to have a greater overall understanding of the monitors' limitations.

The AOP and LTE families reported "no change" in their marriages due to monitoring and expressed an overall "greater comfort" with their monitored infant. The sibling of a SIDS family, however, stated that the monitor was a "cause of a worsening in their relationship" and made their lives "harder with their infant".

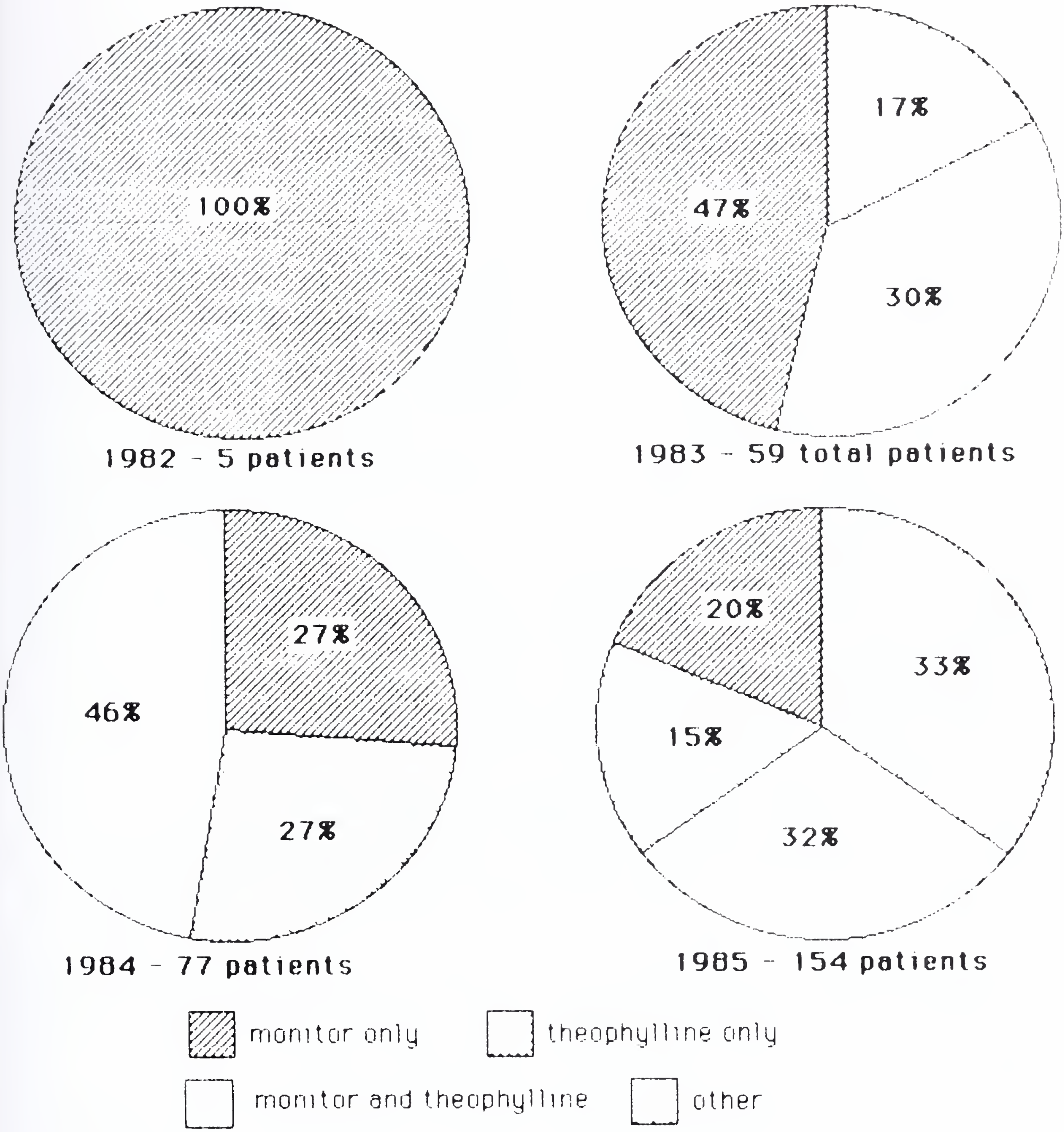
Both the sibling of a SIDS victim and LTE families had greater reluctance and fear in "letting the monitor go". This feeling was not apparent with the AOP family. The AOP family was more assured that apnea was a part of their infant's prematurity, which would resolve with maturity.

In light of our findings, there are several steps being taken in an effort to reduce parental anxiety and establish improved community support systems. During the first week after discharge, referrals are made for monitored families to be visited by a PHN or other health care provider. The visit can focus on assessing areas of need for the family, and should prompt questions such as: 1) does the mother complain of exhaustion or unequal sharing of the monitoring responsibility, 2) are the parents getting out, 3) what are available support systems, and 4) are appropriate mother-infant interactions taking place? It is also an opportunity for well-baby teaching and instilling confidence in parenting skills.

A second home visit is requested during the monitor weaning phase. Those siblings of SIDS and LTE families may especially benefit during the second visit.

Table IV

Treatment modes, 1982-1985



1982 - 5 patients

1983 - 59 total patients

1984 - 77 patients

1985 - 154 patients

While telephone contact is made regularly by the apnea program nurse coordinators, we are restricted to assessing actual monitor concerns.

An infant on an apnea monitor should be attended by a responsible, CPR trained individual who is familiar with and has an adequate understanding of the monitor. Thus, qualified persons with which to leave monitored infants are limited. Comments prompted by the questionnaire encouraged development of lists of qualified sitters and other monitored families for support. To address these needs, the apnea program is organizing a monitored infants babysitter course, a telephone network for monitored families, and monthly monitoring family support group meetings.

Table V

Geographic Locations	1984	1985
Anchorage	46.0%	50%
Kenai Peninsula	16.8%	15%
Matanuska Valley	15.5%	13%
Southeast	5.1%	4%
Fairbanks	1.3%	1%
Other	15.3%	17%

Table VI

Diagnosis	1983	1984	1985
Apnea of prematurity	61%	62%	38.0%
Life threatening event	8%	6%	7.0%
Excessive periodic breathing	8%	4%	21.0%
Gastroesophageal Reflux	8%	5%	4.0%
Obstructive Apnea	6%	—	—
Siblings of SIDS	5%	7%	5.1%
Bronchopulmonary Dysplasia	3%	—	6.4%
Seizures	—	—	3.2%
Parental Anxiety	—	—	1.0%

Summary

The research in infant apnea has not given us concrete answers to the relationship between infantile apnea and SIDS. Testing modes such as polygraphic readings still do not single out all those at risk for SIDS. The Apnea Monitoring Program was developed to provide a means to reduce mortality and morbidity for infantile apnea by improving diagnostic methods, providing follow-up contact with these families, and researching data specific to infantile apnea in Alaska.

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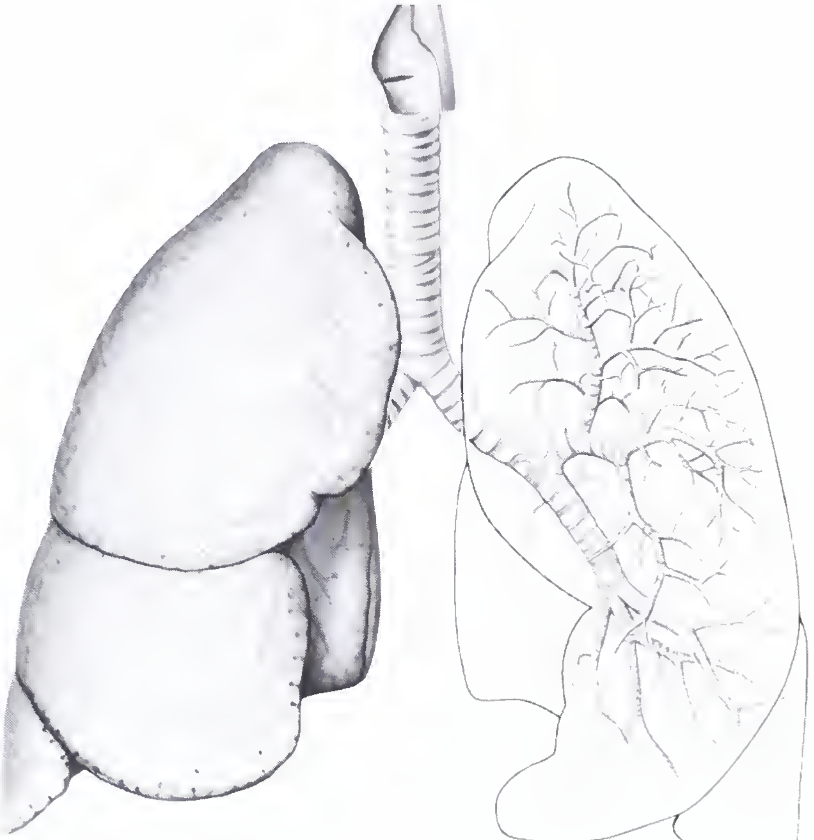
Consider the causative organisms...



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Brief Summary Consult the package literature for prescribing information

Indications and Usage Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae* and *S pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone In moderate to severe cases, management should include sigmoidoscopy appropriate bacteriologic studies and fluid, electrolyte, and protein supplementation When the colitis does not improve after the drug has been discontinued, or when it is severe oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile* Other causes of colitis should be ruled out

Precautions *General Precautions* — If an allergic reaction to Ceclor® (cefaclor Lilly) occurs, the drug should be discontinued and, if necessary the patient should be treated with appropriate agents, e g , pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms Careful observation of the patient is essential If superinfection occurs during therapy appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

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As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy — Pregnancy Category B — Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor® (cefaclor, Lilly) There are, however, no adequate and well-controlled studies in pregnant women Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers — Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses Average levels were 0 18, 0 20, 0 21, and 0 16 mcg/ml at two, three, four, and five hours respectively Trace amounts were detected at one hour The effect on nursing infants is not known Caution should be exercised when Ceclor is administered to a nursing woman

Usage in Children — Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2 5 percent of patients and include diarrhea (1 in 70)

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1 5 percent of patients and include morbilliform eruptions (1 in 100) Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor Such reactions have been reported more frequently in children than in adults Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy No serious sequelae have been reported Antihistamines and corticosteroids appear to enhance resolution of the syndrome

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients)

Causal Relationship Uncertain — Transitory abnormalities in clinical laboratory test results have been reported Although they were of uncertain etiology they are listed below to serve as alerting information for the physician

Hepatic — Slight elevations in SGOT SGPT, or alkaline phosphatase values (1 in 40)

Hematopoietic — Transient fluctuations in leukocyte count predominantly lymphocytosis occurring in infants and young children (1 in 40)

Renal — Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200)

[061782R]

Note Ceclor® (cefaclor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients

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INCEST IN ALASKA: A LEGAL AND ETHICAL DILEMMA FOR THE PHYSICIAN

David J. Coons, M.D.
Malgosia Cegielski, Ph.D.

Abstract:

Incest is a wide spread but underreported phenomenon which has recently come to greater public attention. The physician is often the first to identify incest cases. Alaska's 1982 attempt to deal with incest through changes in the law has placed the health care provider who discovers an incest case in a complex and difficult ethical dilemma. The implications of the current legal situation in Alaska relative to incest are explored. A brief history of the study of incest, some of the dynamics of the incest process, and the identified effects of incest on victims are also outlined.

Historical Perspective:

References to incest have existed throughout recorded history where the topic has been the problem of religious and moral thinkers and thus, the law. Historically, incest has also been a subject of speculation and theoretical interest in the social sciences, but the considerable body of existing literature reveals few well designed studies (1). Almost all studies are based on reports of child protection agencies, mental health care delivery systems, or the court, thus, overrepresenting the most disorganized and least sophisticated family units. In recent years, authorities have come to believe that the incidence of incest is much greater than was once thought (2).

Epidemiology:

Masters and Johnson, report in 1976 that even today society tends to deny or minimize the reality of sexual abuse of minors by family members (3,4). In addition to denial, incest is generally believed to be very underreported. Some authorities believe that there is an epidemic increase in the actual incidence of incest while others believe child sexual abuse cases are simply coming to greater public attention (5,2). Contrary to the popular assumption that incest occurs only in the defective or degenerate members of society, it in fact exists in all social strata (6). Unfrotunately accurate incident statistics do not exist. One conservative estimate based on a report of the National Center on Child Abuse and Neglect is that a minumum of 100,000 cases of abuse occur annually in the United States. It is estimated that 50 to 90% of all sexual assaults on children go unreported and that 70 to 80% of the cases are incestuous in nature (7). In Alaska incest accounts for 75% of all sexual abuse cases reproted (8). In the vast majority of cases the offender is an adult male and the victim, a minor female. This is most commonly father/daughter incest with father /step-daughter being next in frequency. Although subling incest is also quite common it is even less reported and has not been the subject of as much interest, study, or concern. Because of the limited data on other forms of incest this paper will focus primarily on father/daughter and father/step-daughter incest.

Daughter

The victim finds herself in a precarious situation which makes disclosure of the sexual abuse secret difficult. Frequently a close emotional relationship exists between the offender and the victim with reproting unlikely since the adult is in the position of a trusted caregiver to a prepubescent child. The perpetrator rarely uses physical force but rather uses his position of inherent power over the victim. Because there is a superficial impression that the victim consents to the sexual abuse she is often blamed, at least in part, for a situation over which she has no control. Once the situation is revealed, society at large, the perpetrator, other family members , and the legal system may directly or indirectly blame the victim. While more sophisticated authors do place the onus of responsibility with the perpetrator (9), there is a body of literature which has pointed to the contributions of the victim to her own situation (9) and even some that takes on an accusatory tone. However, the blame conveyed by others is generally much smaller than what the victim places on her self (6). This self-blame can occur in the youngest of victims or in the most abusively extreme circumstances. The victim typically feels responsible for both the incest itself and subsequent disruption of the family when the secret is disclosed. Central to the incest relationship is the fact that children are, by nature, dependent on adults for both their physical and emotional needs. Likewise, the child depends on the parents to teach her about appropriate ways to express and meet social and emotional needs since she has no inate way of knowing how to handle physical arousal or sexual behavior. Even when a child knows that something is wrong, the need for attention and a "special" relationship can keep the child participating in the incest. All too often the sexual contact is the only form of warmth or closeness that the child experiences. While healthy parental relationships are directed toward educating and aiding the child in managing emerging sexuality, the incest process short-circuits development through a grave betrayal of trust (10).

Effects on the Victim:

Most researchers agree that the victim almost always suffers significant adverse effects from sexual abuse (4). Short-term emotions experienced during the incest and/or shortly after its disclosure include fear, guilt, humiliation, feelings of low self-esteem, anxiety, confusion, and the feeling of being betrayed or used. Feelings of betrayal extend not only to the perpetrator who has failed to follow established boundaries, but also to the mother who has failed to function in a protective capacity. Often the non-offending parent "does not see" cues that the sexual abuse is occurring or does not believe the child who finally discloses the incest. Besides emotional effects actual behavior changes such as anti-social behavior, school avoidance, withdrawl, bedwetting, fear of being alone, fear of the dark, sexual phobias, and sexual promiscuity are commonly manifested by the sexually abused child. Some of these behaviors and feelings are unresolved well into adult life. As adults, victims often have impaired relationships, sexual dysfunction or repeatedly enter into relationships where they are victimized.

Reporting Requirements:

Every state mandates the reporting of child abuse, including sexual abuse. This requirement takes precedence over medical confidentiality. Nonetheless, it is widely recognized that professionals tend to underreport abuse cases for a variety of reasons (7,11).

The Alaska Law:

In 1981, legislation was introduced to stiffen penalties for sexual assault. This legislation includes a section which classifies incest cases which involve penal penetration as a category of sexual assault in the first degree, thus requiring an eight year mandatory sentence for one convicted of the crime (AS12.55). Although the law was intended to deter offenders, it presents some major problems to anyone who becomes aware of an incest case, and for the incest victim and her family, as well.

Negative Implications Of A Mandatory Sentencing Law:

The law provides some disincentive to reporting because of its severity. There is considerable difference for the victim, the perpetrator, or the non-offending parent in reporting sexual abuse for which consequences require limited prison sentences and probation and treatment versus consequences that will remove a husband, father and provider from the home for eight years. This problem is most apparent when the perpetrator feels guilty and wants to seek help on his own. The law prosecutes this individual in the same way as a perpetrator who dinies the sexual abuse, leaving the judge no choice in sentencing. However, evidence is accumulating that the offender who seeks help on his won and is willing to take responsibility for his actions is most likely to benefit from treatment. Mandatory sentencing can have delitarious effects on the victim and on her family as well. When the victim begins to assert increasing objection to the incest situation and makes moves toward disclosure only to discover that this will result in an extended prison sentence for her father, she may be more reluctant to disclose.

Almost without exception, victims of incest experience guilt not only from the sexual abuse, but from the disclosure when it occurs (6). There is often considerable anger toward the victim from other family members which can result in her emotional isolation. Financial hardship often looms over the victim and her family when the perpetrator is the primary wage earner. At times these pressures become so great that she will retract her accusation (10). Further compounding her isolation is the fact that it is the victim who is often removed from the home as a first step by the authorities, rather than removing the perpetrator (10). Finally, she is repeatedly questioned about a very uncomfortable subject matter by various personnel from service providing agencies, the court, and the law enforcement system. She may later find that a primary motive for their involvement is sentencing the perpetrator, a fact that becomes especially clear if she decides to retract her story. By the time therapy is initiated, the victim has frequently gained a significant distrust for those who offer "help" and she feels very isolated.

A mandatory sentencing law also has indirect effects on the victim. Under other systems which require reporting but have flexible sentencing, it is often in the best interest of the perpetrator to admit his guilt, avoid a trial, and hope for the best. Under mandatory sentencing, it makes much more sense for the perpetrator to force the case to trial since there is a fair chance that the victim will retract or alter her story or the jury will choose not to convict. This is especially so since the judicial system in these situations is particularly hard on children who can be poor wintesses and easily manipulated by the defense. This produces a bizarre paradox where potentially more treatable perpetrators who experience guilt and would confess, may suffer graver consequences than the pernicious abusers who lie, manipulate, and fight with a fair chance of avoiding punishment or treatment.

Treatment Alternatives:

While it is clear that some perpetrators require extended prison time to protect the victim or society, prison time in and of itself is in no manner rehabilitative nor can it be considered treatment. There are many factors to consider in making treatment decisions, and even when the perpetrator can not return home often he could still be a productive member of society following a sentence much shorter than Alaska's eight year presumptive sentence (12). Statistics vary regarding recedivism rates, but a significant number of perpetrators can be returned to the home with ongoing treatment and case monitoring (12). The literature repeatedly points out that the incest process is a complex family interaction which involves not only the victim(s), but the rest of the family (6). Failure to treat the entire family system leaves the family members and the victim open to further exploitation even in the absence of the perpetrator.

It has become quite clear that it is necessary to involve the criminal justice system in dealing with incest. In the past, attempts at treatment without the strong incentive of prosecution and/or prison have often failed. The perpetrator will try to involve his therapist in a power struggle which he can win in the absence of the legal system. The most successful programs involve close cooperation between the courts, criminal justice system, and treatment providers. Then, cases can be dealt with as rapidly as possible with the goal of mandating treatment under a tightly structured probation. Attempts are made to avoid a full trial and to eventually reunite the family, if possible. However, priority is always placed on protection of the victim and there can be little doubt that many cases must begin with specialized inpatient treatment or incarceration. Out-patient case management is dealt with cautiously and with regular and frequent probationary monitoring. Even at the early stages, the well being of the victim should be a primary consideration, e.g. she is the one who stays and the abuser is the one who leaves the home until it is determined if it is safe for him to return.

The Physicians Role:

Along with school personnel, the physician is likely to be one of the first to become aware of an incest case (7,11). Research shows that physicians tend to have low sensitivity to the possibility of incest even in those cases where it should be strongly suspected. There has been a tendency to avoid asking even the most basic questions related to sexual abuse and cases of dropping victims from treatment when abuse is suspected have been documented. Medical records are often medicolegally inadequate (13).

Overlooking or ignoring incest is not a suprising phenomenon, since even those who regularly work with abused children find it difficult to deal with incest. The physician has not been well prepared to confront any child abuse. Reporting patients to the authorities seems like the antithesis of the caring and protective role which the physician is taught. Incest is a particularly uncomfortable aspect of practice which the physician is not prepared to confront. Compounding the problem is the fact that physicians are used to working alone or in a strictly medical grouping and involvement in the icest case brings forth a whole series of contacts with agencies and people who not only take control of the case, but may, at times, seem destructive to everyone involved. Finally, not only the family, but often the victim can become angry at the doctor for following his or her legal duty to report.

Despite all the drawbacks, it remains imperative that incest be reported both for the victim's and society's benefit, and because it is a clear legal requirement. Likewise, the physician is obligated to investigate adequately when presented with physical conditions which are frequently associated with sexual abuse (vaginal or anal discharge, genital trauma, pregnancy, etc.), (13). The report will precipitate a crisis in the family and physicians should be prepared to refer the family members to other professionals who can conduct appropriate therapy. Most physicians have neither the desire nor the training to do the treatment themselves and the act of reporting will generally preclude them from a direct therapeutic role. Failure to report implies complicity with the offender. The threat of reporting, "If this happens again, I'll report you," is not an effective form of blackmail.

Discussion:

The physician, along with other care providers in Alaska, is currently caught in a severe bind by the provisions of the Alaska law concerning sexual abuse. Just as there was growing awareness of the need for increased sensitivity to child sexual abuse a law was passed that makes this awareness a source of potential grief. The current form of intervention can be destructive to the family and victim in the zeal to punish the perpetrator. Incest more than any other crime involves a "love/hate" relationship which places all involved in a position of vulnerability unduplicated elsewhere. Cases range from sadistic terrorism to very complex, but loving relationships which have gone wrong. It is not always a situation to be treated with a sledge hammer as it often is. The physician does not want to be in a position of causing harm, but often it seems there is currently in Alaska no harmless route to take.

Hopefully, Alaska can consider some of the more innovative solutions to dealing with incest that have been developed elsewhere. No one wants to return to the completely inadequate levels of intervention that existed before the change in the law. A system which would allow cooperative intervention by the courts, child welfare agencies, physicians, treatment programs, and the correctional and probation system would however be a huge advance that could prevent the permanent disruption of certain families, yet protect the victim. If there ever was a place for "victim oriented law", this is it. The physician with his or her knowledge of the complexities of the family response to trauma can not only initiate intervention but help smooth some of the crisis atmosphere which develops. The current law limits the ability to help the family and puts the physician in a very difficult spot. Awareness of the problem may in the future, allow some solutions to this dilemma.

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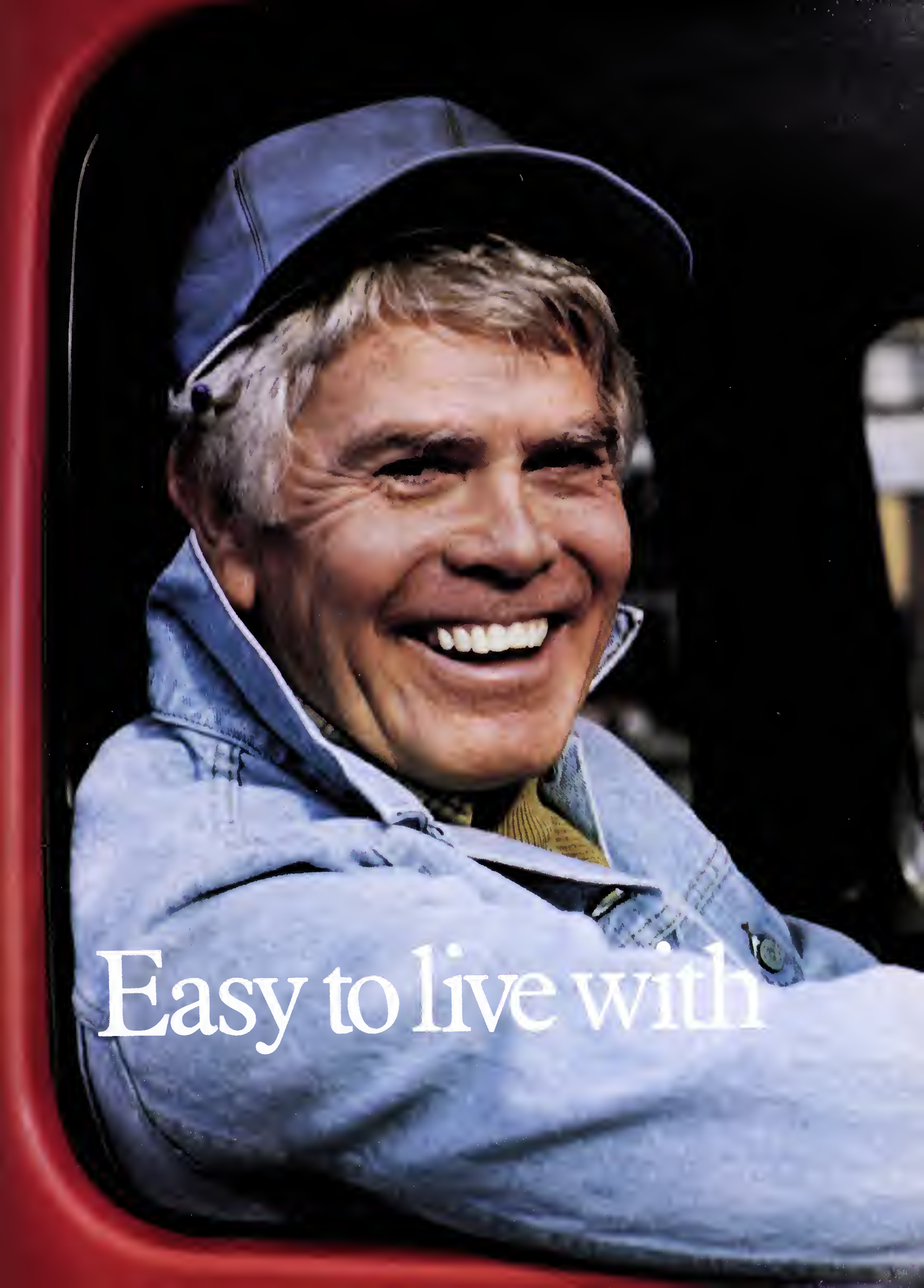
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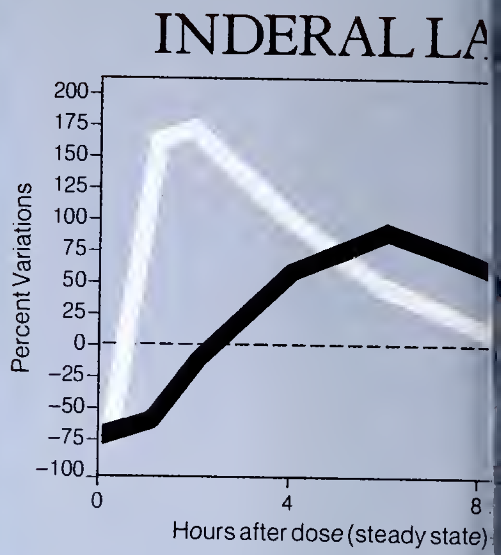

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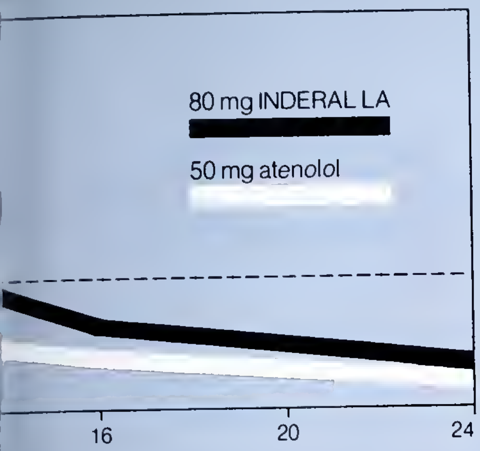

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Propranolol hydrochloride (INDERAL® LA): Propranolol is contraindicated in. 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with propranolol.

Hydrochlorothiazide: Hydrochlorothiazide is contraindicated in patients with anuria or hypersensitivity to this or other sulfonamide-derived drugs

WARNINGS

Propranolol hydrochloride (INDERAL® LA): CARDIAC FAILURE. Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated, and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or propranolol should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction following *abrupt* discontinuance of propranolol therapy Therefore, when discontinuance of propranolol is planned the dosage should be gradually reduced and the patient carefully monitored In addition, when propranolol is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice If propranolol therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute propranolol therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol.

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD, IN GENERAL, NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution, since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin. Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure

Hydrochlorothiazide: Thiazides should be used with caution in severe renal disease. In patients with renal disease, thiazides may precipitate azotemia. In patients with impaired renal function, cumulative effects of the drug may develop

Thiazides should also be used with caution in patients with impaired hepatic function or progressive liver disease, since minor alterations of fluid and electrolyte balance may precipitate hepatic coma

Thiazides may add to or potentiate the action of other antihypertensive drugs. Potentiation occurs with ganglionic or peripheral adrenergic-blocking drugs

Sensitivity reactions may occur in patients with a history of allergy or bronchial asthma

The possibility of exacerbation or activation of systemic lupus erythematosus has been reported

PRECAUTIONS

Propranolol hydrochloride (INDERAL® LA): GENERAL Propranolol should be used with caution in patients with impaired hepatic or renal function Propranolol is not indicated for the treatment of hypertensive emergencies

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that propranolol may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs, such as reserpine should be closely observed if propranolol is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity, which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies, in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

PREGNANCY Pregnancy Category C. Propranolol has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximal recommended human dose. There are no adequate and well-controlled studies in pregnant women. Propranolol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS Propranolol is excreted in human milk Caution should be exercised when propranolol is administered to a nursing mother.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

Hydrochlorothiazide: GENERAL. Periodic determination of serum electrolytes to detect possible electrolyte imbalance should be performed at appropriate intervals.

All patients receiving thiazide therapy should be observed for clinical signs of fluid or electrolyte imbalance, namely: Hyponatremia, hypochloremic alkalosis, and hypokalemia Serum and urine electrolyte determinations are particularly important when the patient is vomiting excessively or receiving parenteral fluids. Medication such as digitalis may also influence serum electrolytes Warning signs irrespective of cause are. Dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, and gastrointestinal disturbances such as nausea and vomiting

Hypokalemia may develop, especially with brisk diuresis, when severe cirrhosis is present, or during concomitant use of corticosteroids or ACTH

Interference with adequate oral electrolyte intake will also contribute to hypokalemia. Hypokalemia can sensitize or exaggerate the response of the heart to the toxic effect of digitalis (eg, increased ventricular irritability). Hypokalemia may be avoided or treated by use of potassium supplements, such as foods with a high potassium content

Any chloride deficit is generally mild and usually does not require specific treatment, except under extraordinary circumstances (as in liver or renal disease). Dilutional hyponatremia may occur in edematous patients in hot weather; appropriate therapy is water restriction, rather than administration of salt, except in rare instances when the hyponatremia is life-threatening. In actual salt depletion, appropriate replacement is the therapy of choice.

Hyperuricemia may occur or frank gout may be precipitated in certain patients receiving thiazide therapy

Insulin requirements in diabetic patients may be increased, decreased, or unchanged Diabetes mellitus which has been latent may become manifest during thiazide administration.

If progressive renal impairment becomes evident, consider withholding or discontinuing diuretic therapy

Thiazides may decrease serum PBI levels without signs of thyroid disturbance.

Calcium excretion is decreased by thiazides. Pathologic changes in the parathyroid gland with hypercalcemia and hypophosphatemia have been observed in a few patients on prolonged thiazide therapy The common complications of hyperparathyroidism, such as renal lithiasis, bone resorption, and peptic ulceration, have not been seen. Thiazides should be discontinued before carrying out tests for parathyroid function

DRUG INTERACTIONS Thiazide drugs may increase the responsiveness to tubocurarine.

The antihypertensive effects of thiazides may be enhanced in the postsympathectomy patient. Thiazides may decrease arterial responsiveness to norepinephrine. This diminution is not sufficient to preclude effectiveness of the pressor agent for therapeutic use.

PREGNANCY: Pregnancy Category C. Thiazides cross the placental barrier and appear in cord blood The use of thiazides in pregnancy requires that the anticipated benefit be weighed against possible hazards to the fetus These hazards include fetal or neonatal jaundice, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult.

NURSING MOTHERS Thiazides appear in human milk. If use of the drug is deemed essential, the patient should stop nursing

PEDIATRIC USE Safety and effectiveness in children have not been established

ADVERSE REACTIONS

Propranolol hydrochloride (INDERAL® LA): Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular: Bradycardia, congestive heart failure; intensification of AV block, hypotension; paresthesia of hands; thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System Lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability; slightly clouded sensorium; and decreased performance on neuropsychometrics

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation; mesenteric arterial thrombosis; ischemic colitis.

Allergic Pharyngitis and agranulocytosis; erythematous rash; fever combined with aching and sore throat; laryngospasm and respiratory distress.

Respiratory: Bronchospasm

Hematologic: Agranulocytosis; nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes; dry eyes; male impotence; and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes, and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

Hydrochlorothiazide:

Gastrointestinal. Anorexia, gastric irritation, nausea, vomiting, cramping, diarrhea, constipation; jaundice (intrahepatic cholestatic jaundice), pancreatitis, sialadenitis.

Central Nervous System. Dizziness, vertigo, paresthesias; headache; xanthopsia

Hematologic: Leukopenia, agranulocytosis; thrombocytopenia; aplastic anemia

Cardiovascular Orthostatic hypotension (may be aggravated by alcohol, barbiturates, or narcotics).

Hypersensitivity: Purpura, photosensitivity; rash; urticaria, necrotizing angiitis (vasculitis, cutaneous vasculitis); fever, respiratory distress, including pneumonitis; anaphylactic reactions.

Other. Hyperglycemia, glycosuria, hyperuricemia, muscle spasm; weakness; restlessness; transient blurred vision

Whenever adverse reactions are moderate or severe, thiazide dosage should be reduced or therapy withdrawn.

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CANCER IN ALASKAN NATIVES: A 15-YEAR SUMMARY

Anne P. Lanier, M.D., M.P.H.

Lisa R. Knutson, M.S.

Abstract

This study presents an overview of data on cancer incidence in Alaskan Natives for the 15-year period 1969-83. We identified cases of invasive cancer diagnosed during this period in Alaskan Natives through review of hospital records and death certificates. The most frequently diagnosed cancers in this population include colorectal, lung and breast cancer. After adjustment for differing age structures, Alaskan Natives had an overall cancer incidence rate similar to that of U.S. whites. However, the populations differed in rates of cancer at specific sites. Over the 15-year study period, the rates of all cancers combined, and specifically lung cancer, increased. We plan further study of these data by geographic region and ethnic group.

Introduction

In recent years Alaskan Natives have expressed considerable concern over the apparent increase of cancer in their population. This observation is particularly significant since, until fairly recently, cancer was thought to be rare among Eskimos and other Native groups. Although reports of cancer in Alaskan Natives have been previously published(1-10), collection of cancer incidence data for Alaskan Natives for 1969-83 has just been completed. This report presents some of our findings. More detailed analyses by region, ethnic group and site are planned.

Methods

All Alaskan Natives (Eskimos, Indians, Aleuts) and American Indians who developed cancer during 1969-83 and were residents of Alaska at the time of diagnosis were identified by review of in-state hospital tumor registries (when available), hospital discharge diagnoses, surgical pathology and autopsy reports, and death certificates. Alaskan Native patients were aslo identified through the Seattle area Cancer Surveillance System. Only data on demographic characteristics, primary site and histologic type of cancer, date of diagnosis, confirmation of diagnosis and main treatment facility were collected. Data were not gathered on stage at diagnosis, treatment or follow-up. Data collected conformed to the National Cancer Institute's Surveillance Epidemiology and End Results (SEER) program to allow comparison with rates published for other populations. Except for melanoma, cancers of the skin were not registered. In this report only invasive cancers are reviewed.

Population figures used in calculations are from the 1970 and 1980 censuses. The numbers of cancers diagnosed (observed) were compared with a calculated number (expected) based on the age and sex distribution of the Alaskan Native population and published cancer incidence rates (Third National Cancer Survey) for U.S. whites. Significant values of the observed to expected ratios (O/E) were identified using the Poisson distribution. In addition, to further facilitate comparisons, cancer incidence rates for Alaskan Natives were age-adjusted to the 1970 U.S. population.

To examine trends, the total Native cancer experience over the 15-year study period was determined for all cancers and for each site. From this information, the expected annual number of cases was determined, taking into consideration both increases in total population and changes in age distribution. The difference between this number and the number of patients actually diagnosed each year was then determined. The data are presented as three-year moving averages.

An estimate of the Native population at risk for cancer in the year 2000 was obtained by using 1980 census data and death rates for non-whites in the U.S. The projection was based only on those

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persons alive in 1980, so no estimates were made for persons under age 20 in the year 2000. Persons in this age group account for only a small proportion of the total number of cancers in a population.

Results and discussion

The numbers of invasive cancers identified during 1969-83 and the age-adjusted cancer incidence rates are shown by site and sex in Table 1. Our survey is as complete and accurate as possible, since 1) most Alaskan Natives receive their health care directly or indirectly through the Indian Health Service system, 2) multiple resources, including Seattle area facilities, were surveyed to identify cases, and 3) death certificates were also reviewed.

Table 1
Alaskan Natives
Numbers of Cases and Rates of Invasive Cancer
1969-83 by Site and Sex

	Number of Cases		Age-Adjusted* Rates Per 100,000 Per Year	
	Men	Women	Men	Women
Colon/Rectum	128	133	61.0	65.2
Lung	158	53	69.7	23.2
Breast	1	115	0.4	44.2
Cervix		83		28.0
Stomach	52	19	22.4	8.4
Prostate	65		34.5	
Kidney	26	27	11.4	11.0
Nasopharynx	35	16	13.1	6.4
Pancreas	24	21	10.1	9.6
Hematologic	29	13	10.7	4.3
Gallbladder	12	29	6.7	14.7
Liver	31	6	10.8	2.0
Oral/Pharynx**	12	24	4.1	9.3
Thyroid	7	26	2.9	6.7
Lymph Node	21	8	8.3	3.1
Ovary		25		9.5
Bladder	18	7	8.9	3.4
Esophagus	18	7	8.9	3.4
GI/Unspec	8	12	3.7	6.2
Bone/Conn. Tissue	12	7	4.6	2.6
Brain	11	7	2.8	1.5
Testis	16		3.7	
Other	20	21	6.3	7.0
Unknown	52	60	24.7	30.9
Total	756	719	329.7	300.6

*Adjusted to U.S. 1970 population.
**Category includes cancers of the lip, oral cavity and pharynx, except nasopharynx which is listed separately.

The ten cancers most frequently diagnosed in Native men and women differ somewhat from those most often diagnosed in U.S. whites (Table 2). Among Native men, nasopharyngeal and liver cancer are among the top ten, while cancer of the larynx and leukemia are not. Among women, cancers of the kidney, gallbladder and thyroid rank among the most frequent, while cancers of the bladder and uterus and leukemia do not. In addition, within the lists of most frequently diagnosed cancers, the relative positions of cancers frequently seen in both populations differ. Cancer of the prostate is diagnosed less frequently than colorectal cancer in Native men, while the reverse is true among U.S. white men. In Native females, colorectal cancer is diagnosed more frequently than breast cancer; the opposite is true for white women.

Table 2
Ten Most Commonly Diagnosed
Invasive Cancers in
Alaskan Natives, and U.S. Whites

Men

Alaskan Natives (1969-83)	U.S. Whites (1983)
1. Lung	1. Lung
2. Colon-Rectum	2. Prostrate
3. Prostate	3. Colon-Rectum
4. Stomach	4. Bladder
5. Nasopharynx	5. Lymphoma
6. Liver	6. Leukemia
7. Kindey	7. Stomach
8. Lymphoma	8. Pancreas
9. Pancreas Esophagus	9. Kidney
10. Bladder	10. Larynx

Women

Alaskan Natives (1969-83)	U.S. Whites (1983)
1. Colon-Rectum	1. Breast
2. Breast	2. Colon-Rectum
3. Cervix	3. Lung
4. Lung	4. Uterus
5. Gallbladder	5. Ovary
6. Kidney	6. Cervix
7. Thyroid	7. Lymphoma
8. Ovary	8. Pancreas
9. Pancreas	9. Leukemia
10. Stomach	10. Bladder

Although the rank order indicates the relative frequency of cancers diagnosed, comparisons with other populations are affected by differences in the age distribution of Natives compared with that of the comparison population. Figure 1 shows the age-specific incidences rates for all cancers in Natives and in U.S. whites. The curves are generally similar.

Before prescribing, see complete prescribing information in SK&F CO. literature or *PDR*. The following is a brief summary.

WARNING

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: **Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired.** If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: The bioavailability of the hydrochlorothiazide component of 'Dyazide' is about 50% of the bioavailability of the single entity. Theoretically, a patient transferred from the single entities of Dyrenium (triamterene, SK&F CO.) and hydrochlorothiazide may show an increase in blood pressure or fluid retention. Similarly, it is also possible that the lesser hydrochlorothiazide bioavailability could lead to increased serum potassium levels. However, extensive clinical experience with 'Dyazide' suggests that these conditions have not been commonly observed in clinical practice. Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Thiazides may add to or potentiate the action of other antihypertensive drugs.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

Supplied: 'Dyazide' is supplied as a red and white capsule, in bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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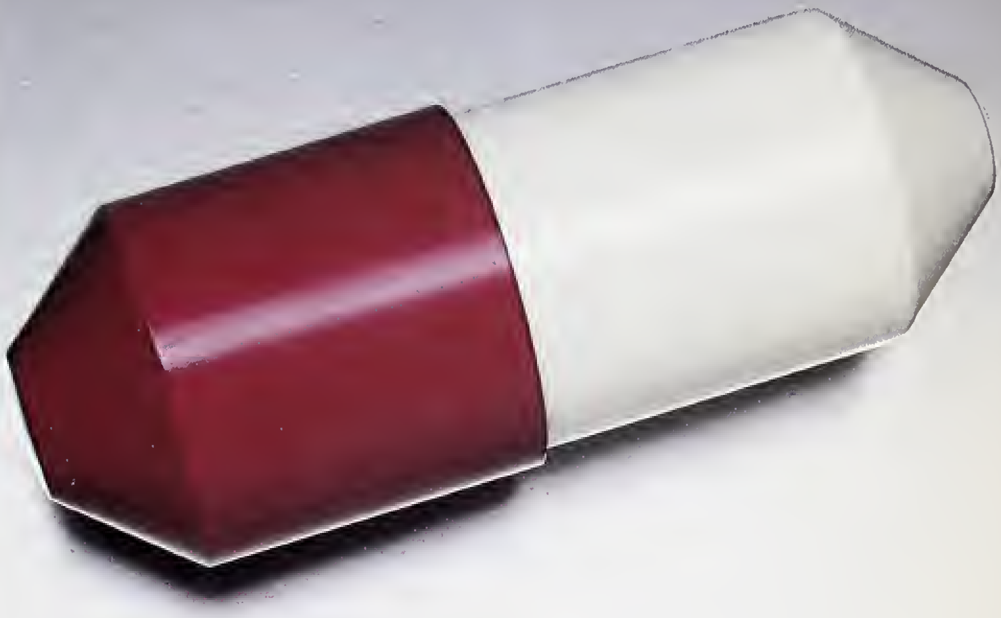

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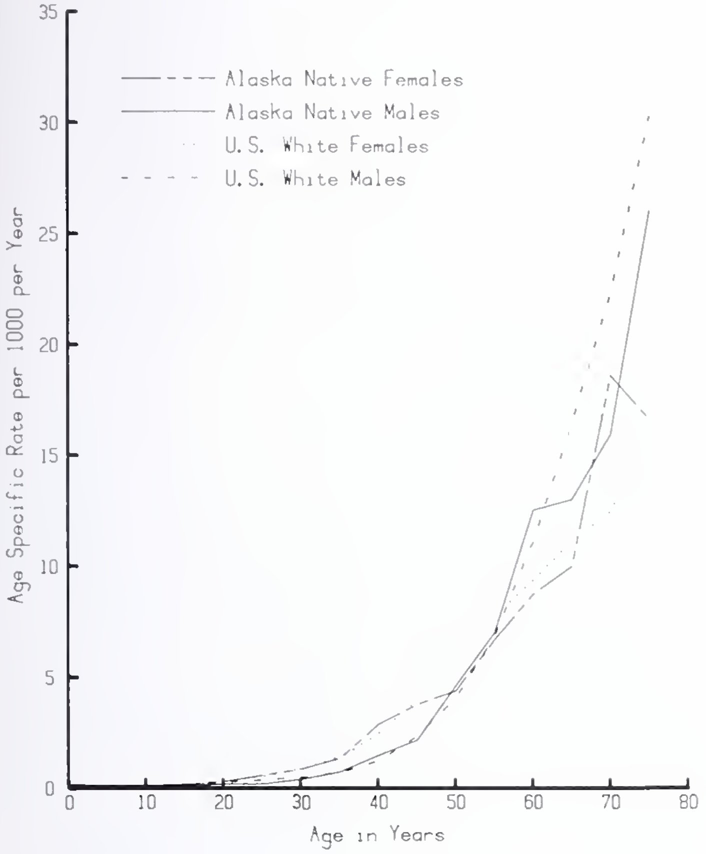
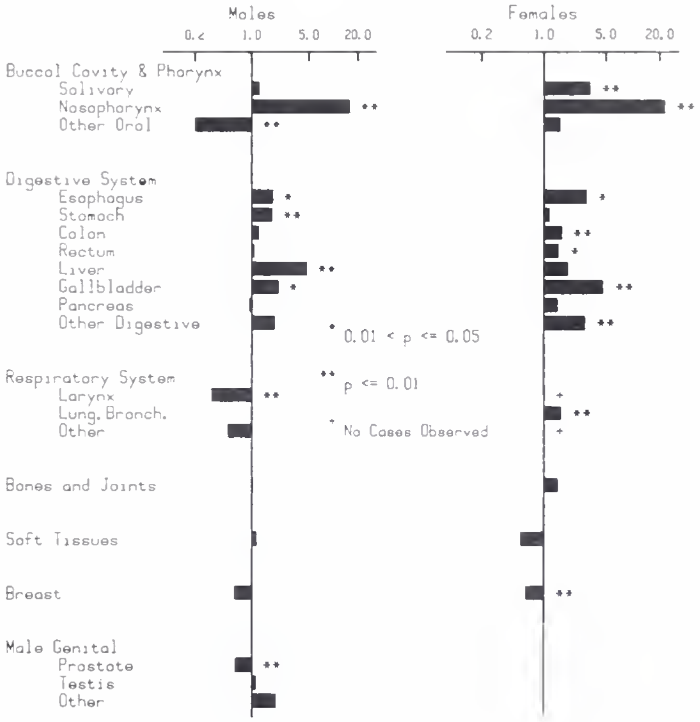


Age-specific incidence rates for invasive cancers: Alaskan Natives (1969-83) compared with U.S. whites (1969-71).



Observed to expected ratios of cancer by site among Alaskan Natives in comparison with U.S. whites.

Figures 2a and 2b show the ratio of cancers observed in Natives compared to cancers expected, based on rates for U.S. whites and Native population figures. Again, the data suggest that, after adjustment for the age distribution of the Native population, the total number of cancers diagnosed among Natives does not differ significantly from that diagnosed among U.S. whites. However, the observed to expected ratios differ markedly for many sites. As shown in Figure 2a and 2b, among Native men or women, and in some instances for both sexes, significant excess cancers are diagnosed in the salivary glands, nasopharynx, esophagus, stomach, colon, rectum, liver, gallbladder, cervix and kidney. On the other hand, in Natives, cancers of the breast, larynx, prostate, uterus, bladder, and nervous system, as well as melanoma, lymphoma, and leukemia, occur relatively less often.

Although 15 years is a short period of time over which to observe trends, data were analyzed for evidence of trends for all cancers and by site. The rate for all cancers increased for both men and women, largely because of significant increases in lung cancer (Figure 3 and 4). Increases, though not significant, were also seen in cancers of the breast and cervix. No downward trends were identified for any cancer site.

Figure 2b

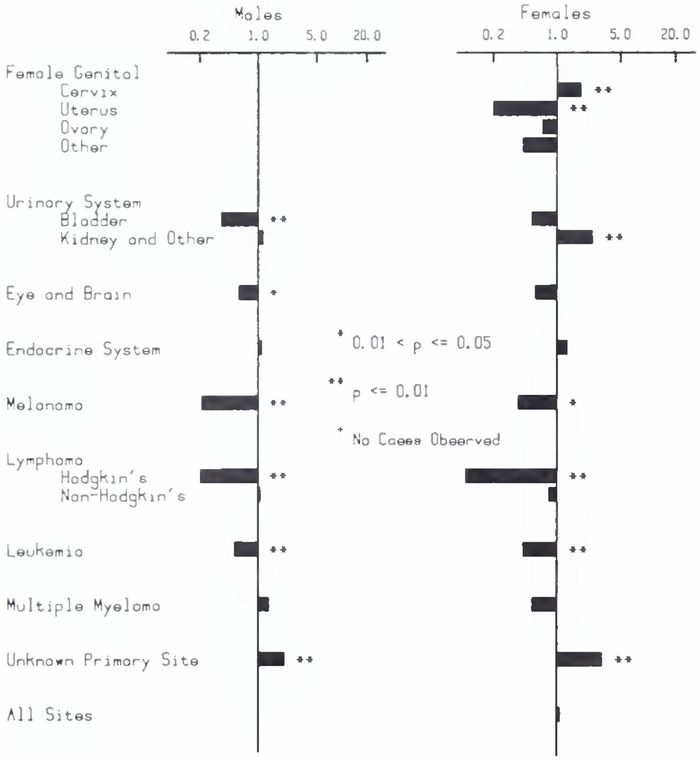
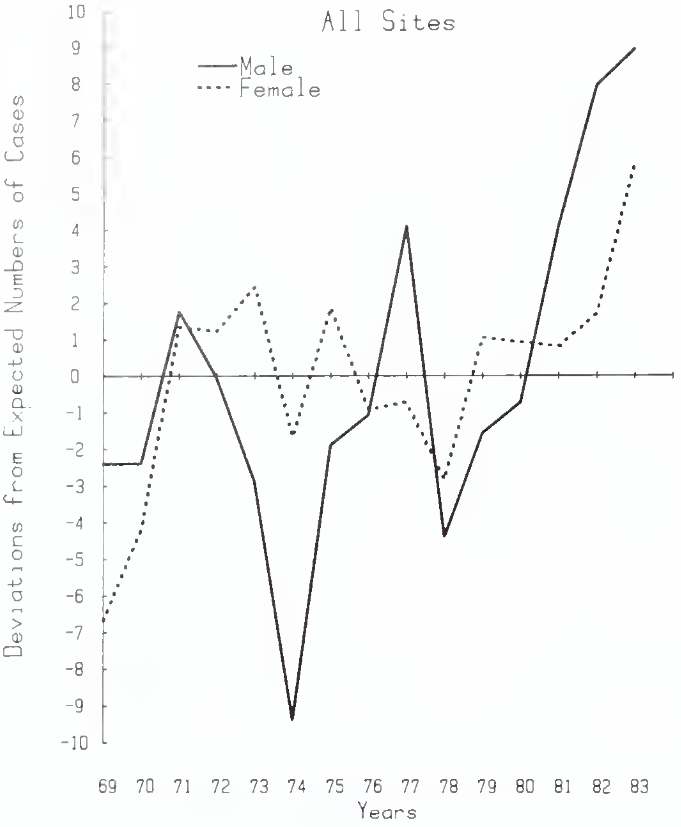
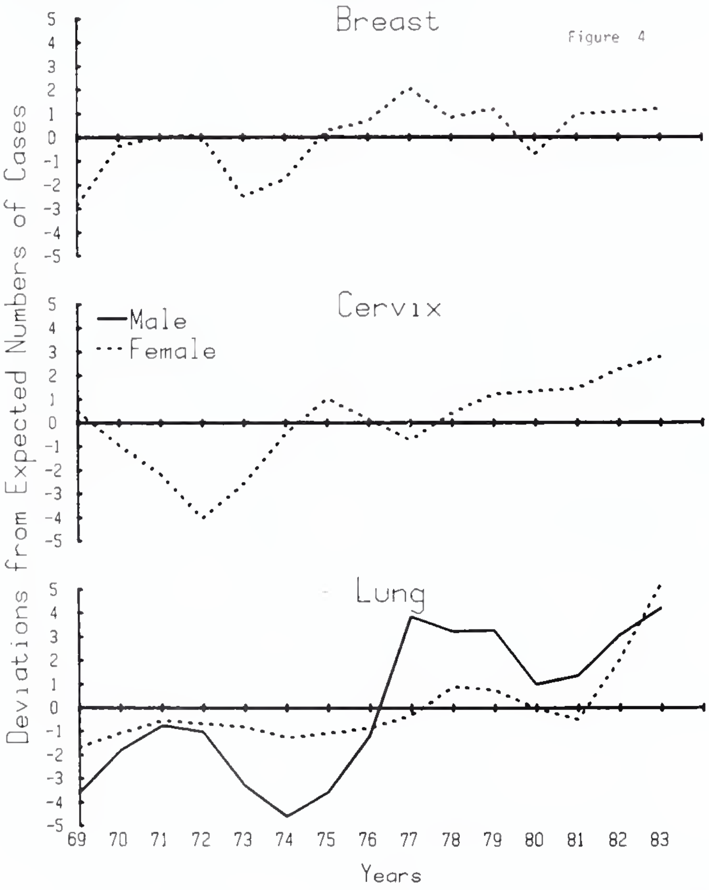


Figure 3



Time trends in cancer incidence among Alaskan Natives for all cancers.



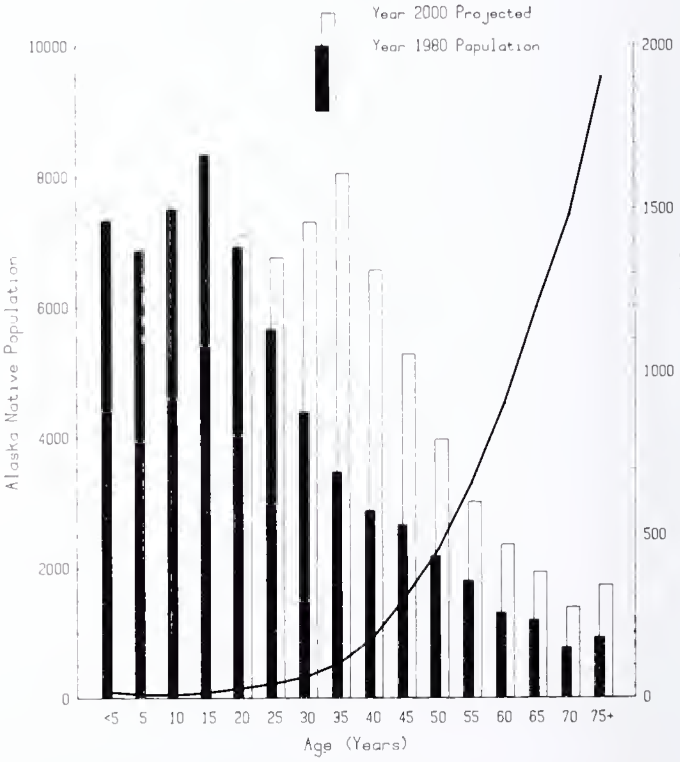
Time trends in cnacer incidence among Alaskan natives for cancers of the breast, cervix and lung.

Conclusion

Data for 1969-83 indicate that after adjustment for age, the cancer rates overall among Alaskan Natives approximate those of U.S. whites, although the types of cancers are significantly different. The number of cancer cases seen over the 15-year period has increased. Although this increase is probably largely a result of increased longevity, with more and more persons entering older age groups, cancer incidence rates for men and women have risen. Much of this increase in incidence rates is due to increases in lung cancer. These data show cancer frequency patterns among Natives are unique, and extrapolation from data on other populations may be inappropriate. Since patterns not only differ but may be changing, current and accurate cancer incidence data must be used to plan programs for treatment, prevention and screening, and for research.

Cancer is already the fourth most frequent cause of in-patient days in the Alaska Area Native Health Service, exceeded only by accidents, deliveries and alcohol.[11] An increase in the number of persons entering the high-risk age groups for cancer is anticipated (Figure 5). Such a population shift can be calculated to result in a doubling over 30 years (e.g., 1980 to 2010) of the number of patients who will develop cancer if rates remain the same. However, since there is evidence of an increase in cancer rates among Alaskan Natives over time and the prevalence of smoking among Natives is known to exceed that in the rest of the United States, the numbers of cancers in natives may exceed those currently predicted.

Figure 5



Projected age distribution of the Alaskan Native population (year 2000) and age-specific cancer incidence rates.

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3363

COST SAVINGS OF REGIONALIZED PERINATAL CARE FOR LOW BIRTHWEIGHT INFANTS: PART I. MORTALITY

Jack Jacob, M.D.

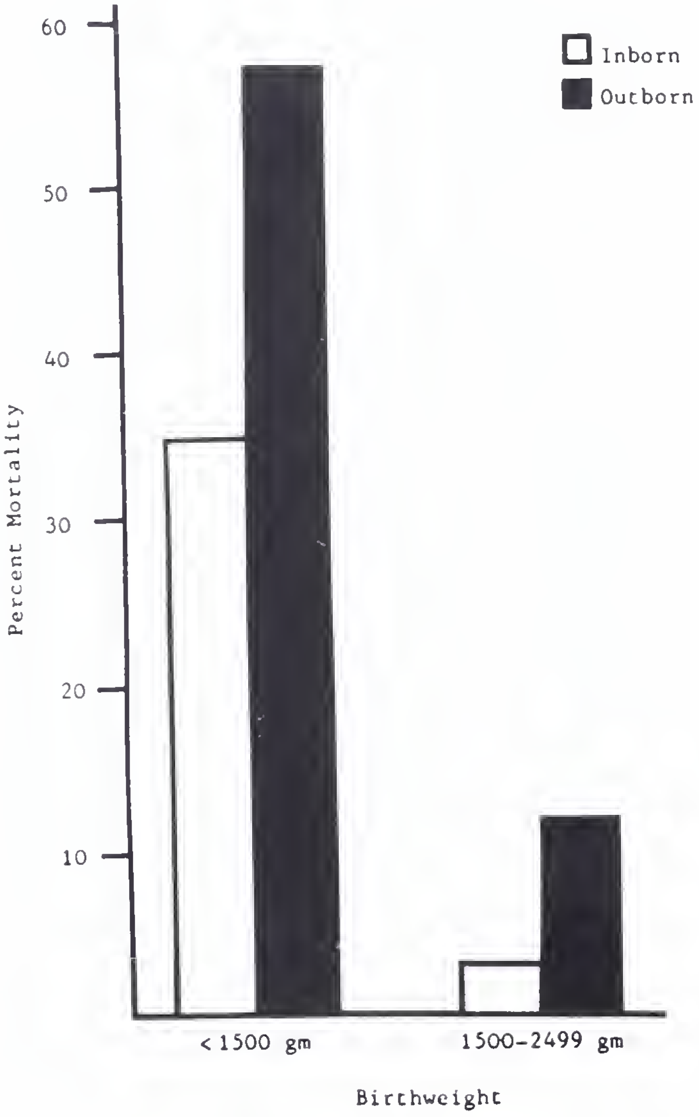
The increased use of neonatal intensive care through regionalization has been responsible for the improved survival of low birthweight infants both in Alaska and elsewhere (1-5). High-risk maternal transport and referral broadens the scope of patients afforded timely newborn intensive care and reduces neonatal mortality and morbidity (6-10). In 1982 we reported neonatal mortality for Alaska could be further reduced by improvements in hospital-perinatal care and increased use of tertiary care perinatal services (1).

The present study was undertaken to assess the impact of the maternal transport and referral program initiated in 1982 in Alaska and its effect on neonatal mortality, cost of hospitalization, and long-term morbidity. Part I of the study places an emphasis on mortality. Part II of this series will describe the effects of regionalization on cost of hospitalization and morbidity.

Methods

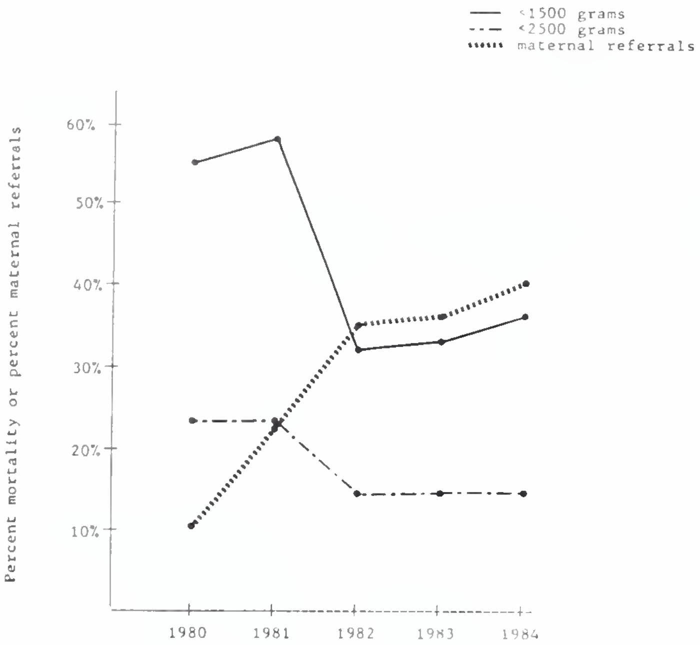
The study was conducted on all low birthweight infants (birthweight ◄ 2500 gm) who were cared for at the Newborn Intensive Care Unit at Providence Hospital from 1980 through 1984. Charts of all infants were reviewed retrospectively. Data extracted consisted of the following: birthweight, race, outcome, place of birth, neonatal vs. maternal transfer, length of tertiary care hospital stay, one and five minute Apgar scores, presence or absence of intraventricular hemorrhage, length on mechanical ventilator, presence of severe chronic lung disease and severe neurological disability.

Figure 1
Comparison of Inborn & Outborn Mortality for LBW Babies: 1980-1984



Clinical Director, Alaska Newborn care Project, 3300 Providence Drive, Suite 05, Anchorage, AK 99508

Figure 2
Mortality Rates According to birthweight Category and its Relationship to Maternal Referrals/Transports



Results

Figure 1 shows a comparison of mortality rates for infants born at the tertiary care center at Providence Hospital and those born outside of this setting. For both birthweight categories mortality is higher for infants born out of the tertiary care setting. Yearly mortality rates for inborn infants varied from 11% to 23% and showed some improvement from 1980 to 1984. Similarly, yearly mortality rates for outborn infants varied from 19% to 37% but showed no significant improvement with time. Figure 2 shows a decline in mortality for LBW infants in 1982 that is correlated with an increase in maternal transport and referral.

Figure 3 shows the decline in mortality for urban and rural areas of Alaska and its relationship to maternal transport and referrals. An infant was placed in the urban category when maternal residence was in the Anchorage or Fairbanks area. All others were placed in the rural category. The reason for this distinction was to separate infants according to ease of access to teritary care. Generally, teritary care is readily available in the communities of Anchorage and Fairbanks but not in any other area of Alaska. The data shows increased acceptance of the concept of maternal transport and referral for the urban population in 1982, and for the rural population in 1983, with simultaneous declines in mortality for both groups. Differences in mortality between urban and rural LBW infants for 1984 were lower than in any previous year.

Figure 4 shows the decline in mortality for native and non-native LBW infants and its relationship to maternal transport and referral. Mortality rates have been decreased for both native and non-native populations.

The acceptance of the concept of maternal transport and referral as an alternative and adjunct to neonatal transport for a certain hospital may be gauged by assessing the fraction of neonatal transports to both neonatal and maternal transports that originate at the hospital. The lower the fraction, the greater the acceptance of maternal transport in the management of the LBW infant. Figure 5 shows trends for acceptance of the concept of maternal transport for years 1982 through 1984 for the three referring hospitals in Anchorage. Figure 6 shows similar data for years 1982 through 1984 combined for infants ◄ 1500 gm birthweight. The data shows a lesser participation in the concept of maternal transport by physicians practicing at Humana Hospital - Alaska for both the low birthweight and very low birthweight infant. Figure 7 shows mortality rates for LBW infants that originated as either neonatal or maternal referrals at the three referring hospitals in Anchorage for the years 1982 through 1984. Higher mortality rates are associated with lesser participation in maternal transport.

Figure 3
Rural & Urban Mortality and its Relationship to Maternal Referrals/Transports for Newborns ◄2500 grams Birthweight

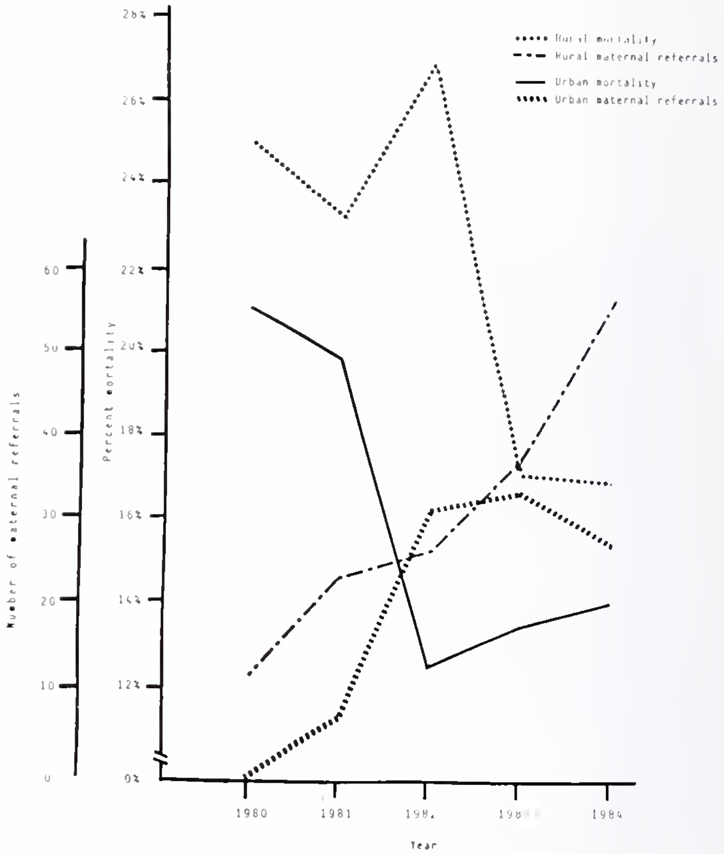


Figure 8 illustrates similar data on acceptance of maternal transports for the population outside the Anchorage area. These data are categorized by race and birthweight. Categorization by hospital was not helpful because of small numbers. The data shows that the only change has been the increased participation by the native population in maternal transport. The degree of participation in

Figure 4
Native and Non-native Mortality and its Relationship to Maternal Referrals/Transports for Newborns ◄ grams Birthweights

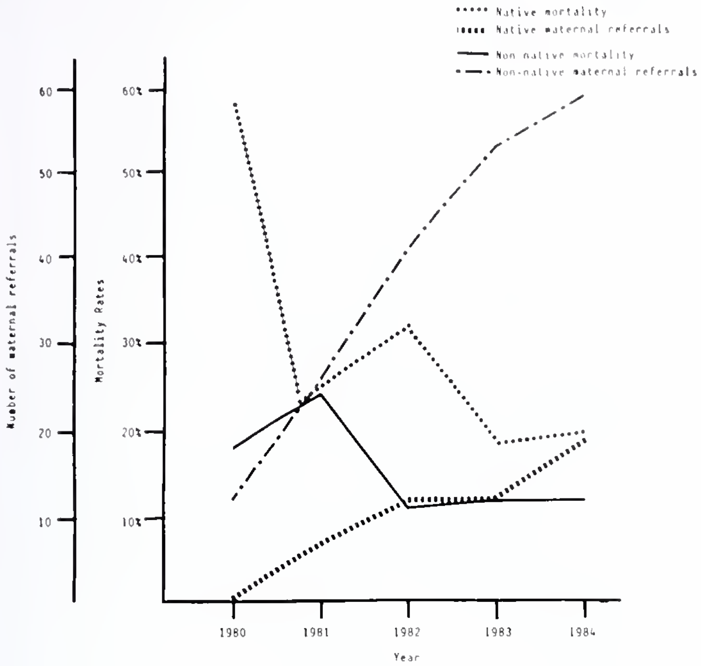
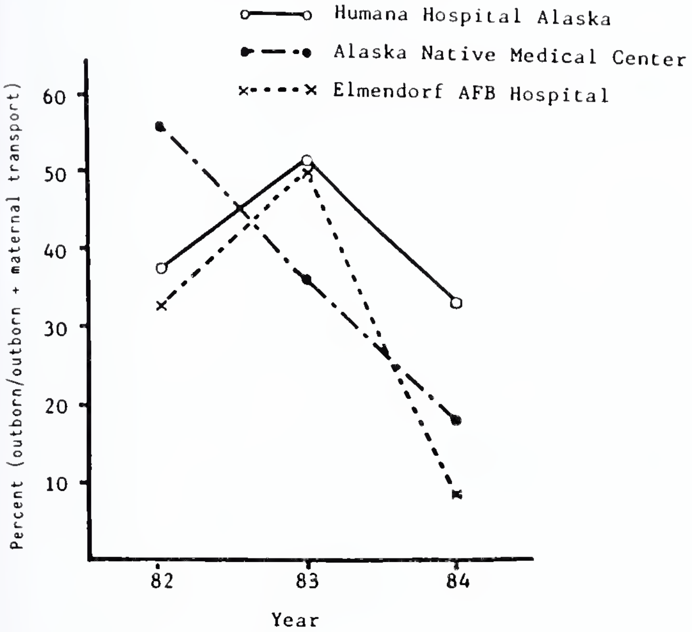


Figure 5
Who participates in perinatal regionalization in the Anchorage area? % outborn infants/total (outborn + maternal transports) Infants ◄1500 grams, 1982-84



maternal transport for infants ◄1500 gm birthweight was similar for native and non-native populations for 1984. The data also shows a greater tendency to resort to neonatal transport for the larger LBW infant.

Discussion

The NICU at Providence Hosptial, as the only tertiary care center in Alaska for high-risk mothers and newborns, cares for a significant proportion of Alaskan LBW infants. The data presented in this paper is therefore felt to be representative of most LBW infants in the state.

Figure 6
Who participates in perinatal regionalization in the Anchorage area? % outborn infants total (ourborn + maternal transports) infants ◄ 1500 grams, 1982-84

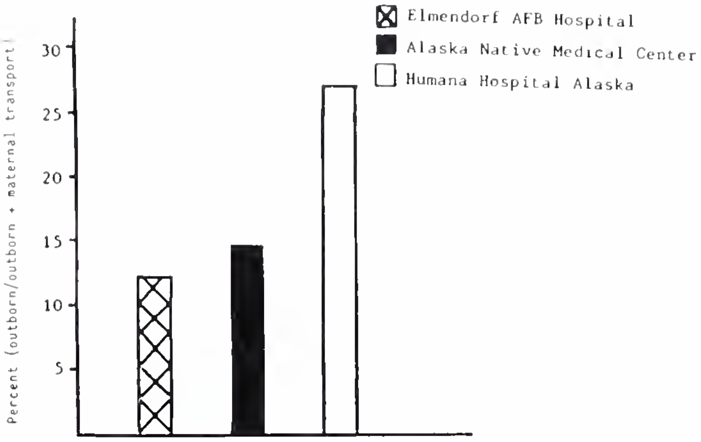


Figure 7
Mortality rates for LBW babies (◄2500 gm) in Anchorage 1982-84

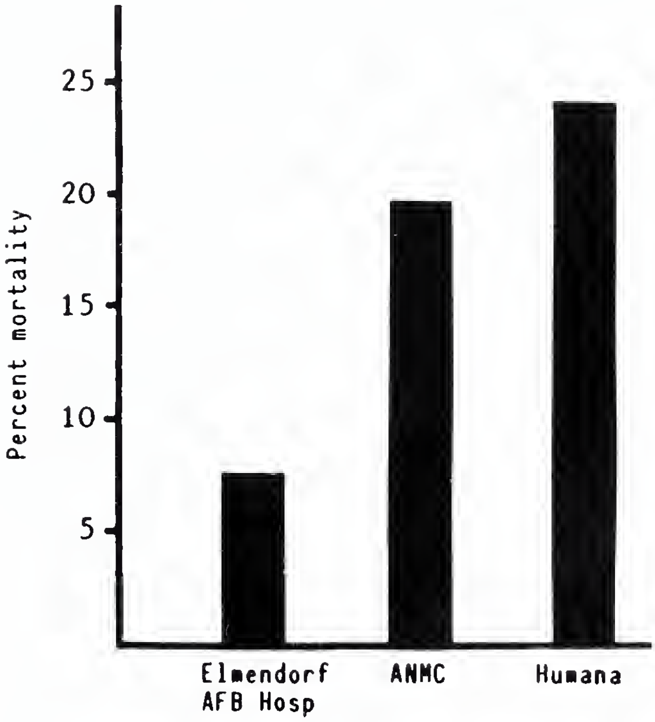
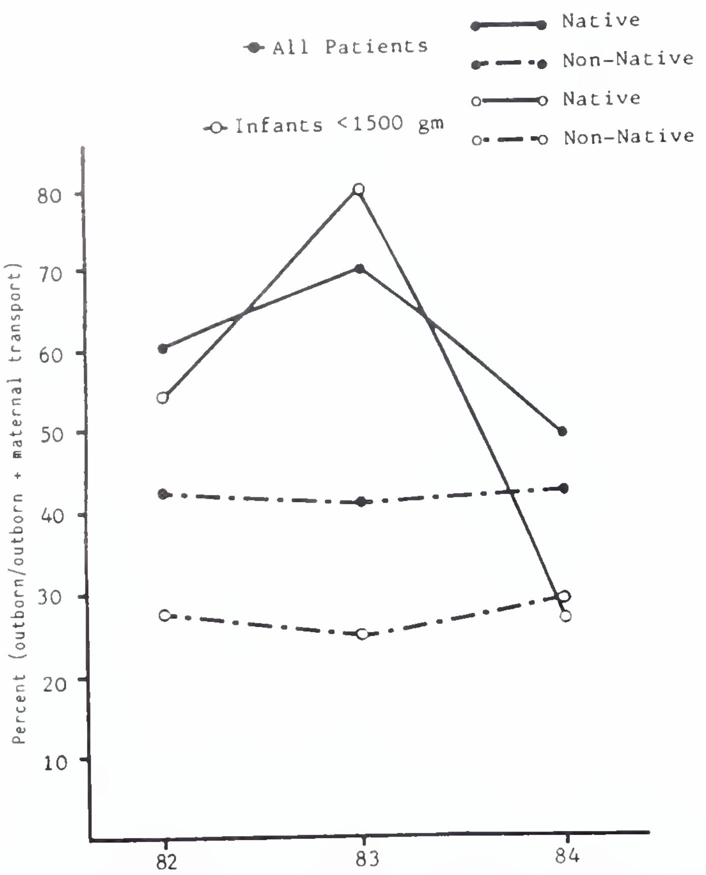


Figure 8
Who participates in regionalization outside the Anchorage area?
Outborn infants/total (outborn + maternal transports)



The study demonstrates an improvement in survival of LBW infants hospitilized at Providence Hosptial between 1980 and 1984 that is principally attributable to the acceptance of the concept of maternal transport and referral in the management of the LBW infant.

In 1982 we suggested (1) that the higher neonatal mortality rates for Alaska natives were concentrated in the native rural population. Similarly, in 1984 the Center for Disease Control published a report (11) documenting a much higher infant mortality rate for Alaska natives, especially those residing in a rural area. Our data shows that maternal transport and referral is a viable solution to equalizing differences in health care delivery for populations that have a higher neonatal mortality rate. In fact, the changes in management of the LBW population has coincided with a major decrease in neonatal morality to 7.8 per thousand live births for Alaska natives for 1984 (12). Furthermore, we feel that the results of this study can serve as a model for other EMS programs in Alaska in order to equalize access to tertiary medical care for rural Alaskans.

Although there are trends toward continued improvement our data point to some problem areas that, if addressed, could make a further impact on reducing neonatal mortality. Within the Anchorage area, where cost of transport is not a significant factor, there are major differences in practice between institutions. Both Alaska Native Medical Center and Elmendorf AFB Hosptial have shown increased participation in maternal referral while Humana Hospital-Alaska has not. Not surprisingly, this has resulted in Humana Hosptial-Alaska having the highest LBW mortality rates for combined neonatal and maternal referrals. This is especially ironic in light of JCAH's designation of Humana Hospital-Alaska as a Level II newborn intensive care center. However, data from New York (5) suggests that LBW mortality in Level II units is no different than in Level I units. This may be due to a greater tendency to keep patients that are more appropriately cared for at a level III center.

There are other trends in health care delivery that will make the task of regionalization and appropriate patient referral more difficult in future years. First, there has been increased competition between hospitals in the private sector in Anchorage. In reality the cooperative relationship between Level II and III hosptials that is the hallmark of regionalized perinatal care (13) does not exist in Anchorage. The problem is worsened by the fact that the traditional "open door" policy that usually allows regional practitioners to participate in delivery of their maternal patients rarely exists at the tertiary care center. In fact, family practitioners in the Anchorage area generally have found their obstetric practice at the tertiary center to be unduly restrictive thereby making the process of maternal referral especially difficult. Finally, insurance programs such as Best Care and Humana Care Plus limit patient and physician choices for medical crea. The goals of such programs may run contrary to the concepts of regionalization. Such programs offer significant financial benefits for the hospital and insurers but may be in conflict with the patients best interest.

The rural sector continues to have lesser numbers of maternal referrals. It is likely that this may be due to unavoidable factors such as women seeking medical care late in premature labor. However, difference between birthweight categories suggests further improvement is possible, especially for the larger LBW infant. Programs directed toward improved education of pregnant women and primary health care providers (including health aides) on the early recognition of complicated pregnancy and premature labor may further improve access to tertiary level perinatal care for rural Alaskans.

Based on the expected outcome of inborn LBW infants for 1984 one can calculate the lives that would be saved if there were greater participation in maternal referrals. If all LBW infants admitted in 1984 were inborn, 9 LBW infants would have been saved. Although this may seem like a small number there are only a total of 60-70 neonatal deaths per year in Alaska making this a significant percentage of neonatal deaths. This would, in effect, decrease the neonatal mortality for Alaska from about 6.5 to 5.5/1000 live births.

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IN MEMORIAM

Abram Cannon, M.D.

A memorial service was held at the First Presbyterian Church, in Fairbanks for Dr. Abram Hoagland Cannon, 70, who died Jan. 12, 1986. The Rev. Keith Wise officiated.

Born Nov. 30, 1915 in Salt Lake City, Utah, Dr. Cannon lived in Fairbanks 14 years. His premedical education was at the University of Utah, a medical degree from Northwestern Univ. Medical School and internship at the Latter Day Saints Hospital in Salt Lake City. He was a retired radiologist and member of the Fairbanks Medical Society, Alaska State Medical Association and American College of Radiology. He served as past president of the Chicago Radiological Society.

In addition, Dr. Cannon was an active member of the New Hope Methodist-Presbyterian Church in North Pole.

Survivors include his wife, Sylvia M. Cannon of North Pole; son, James W. Cannon of Fairbanks; daughters, Margaret Abel of Lewiston, Mont., and Carol Viera of Denver, Colorado and seven grandchildren.

RISK MA

A Short while back, an internist friend gave me some candid feedback concerning the risk management advice I have passed along in Alaska Medicine. "I don't do any of the things you write about," he said. "I've been practicing for thirty years, and I've never yet been sued for malpractice."

Well, one certainly cannot argue with success. With that many years of active practice sans lawsuits, my friend has definitely been successfully managing his risks. I was intrigued. There was no need for an inquiry on my part as to how he achieved this noteworthy result. This physician is known not only for his firmly-held views on a fascinating variety of subjects, but also for his willingness to express those views thoughtfully and forcefully. A complete explanation of the technique began within seconds.

"First," he explained, punctuating his opening point by jabbing an accusing finger in my direction, as if I was part of the problem, "you always write about keeping good records. Well, I never keep the kind of records you suggest. In fact, I deliberately make my records so vague nobody can tell what I am thinking. I don't care if some fancy expert goes over my charts, because they won't make any sense at all to anybody but me."

"And second," he continued, I don't take just anybody for a patient. If they don't have a sense of humor, I don't want them. During the first visit, I make a joke about whatever problem it is they came to see me about. People who are out to sue don't have a sense of humor. For some reason, they get upset with my jokes, and they leave and never come back."

I waited for more, but the lecture was over, almost as soon as it had begun. It was in keeping with most communications the doctor makes: short, often eccentric, and delivered with an air of absolute self-confidence. Actually, there was a third prong of the internist's approach which, with characteristic modesty, he left unstated. I'll state it for him, with an unrequested promise to give equal time for a disclaimer, if he feels it untrue: Those patients who are smart enough to tolerate his odd-ball ways find that they have a doctor who truly cares for them. Day or night, this often gruff, sometimes grouchy, sole-practitioner has let his patients know that he is concerned about them as individuals. Without ever directly explaining what he was doing, he has let his patients know by word and deed that they could place a large portion of the burden of their illnesses on his shoulders. No guarantees of success, of course—but the effective communication of concern is always there.

I most certainly don't advocate this physician's style of record keeping. From my many encounters with medical records, I am firmly convinced that clear, complete, accurate record keeping is an essential part of modern medical practice in the United States. Consider this example: Doctor sees woman, who complains of problem. Doctor diagnoses and treats the problem with medication. During course of treatment, women becomes pregnant. Eighteen years and the period of gestation later, a summons and complaint arrives. The complaint alleges that the prescribed medication caused fetal harm. The once-fetus-now-young-adult is suing for that alleged harm. Is it likely that a doctor will remember the episode giving rise to the complaint? Of course not. Only under the rarest of circumstances would a physician have cause to remember such an incident. Now, however, a mother is going to be crying before a jury, explaining that she had never been told this or that about the medication, and if she had only known whatever some lawyer has been able to show the doctor can't remember, she never would have done what the doctor recommended, and look how her child was damaged for life....

No, I strongly disagree with the internist's chart-keeping techniques. And I don't think I agree with his using trial by sense of humor as a way of eliminating potential patients, either. True, it seems to have been a successful technique for my friend, individually. But what effect might any such technique have on other physicians? Most people hold physicians in extremely high regard. For better or for worse, we occupy a place of high social prominence. If, as a physician I offend a patient, the risk is great that my conduct will be preceived as an example of the arrogance of the medical profession. We are all indicated when the words of one of our members hurts a patient. Offended patients aren't likely to admit their feelings in a place as public as a courtroom—even under oath. They may, therefore, be selected to sit in judgment of a physician. I doubt that many of us would want to be such a doctor.

For all my criticism of the internist's techniques, one thing is clear: He has wandered around in a medicolegal no-man's-land for thirty years without getting shot. Considering the battlefield statistics, that record is considerably better than has been recorded by the occupiers of nearby foxholes.

If I were to identify the physician in question, at least two points would immediately be clear. (I will ask that these be taken on faith, since I wish to preserve the anonymity of the doctor.) First, it is extremely unlikely that his success would be attributed to a well-developed sense of tact and diplomacy. Some would, in fact, marvel at his achievement, which they may joke might have been attained without the exercise of either of those talents. Second, his freedom from litigation probably has not arisen from any particular fondness on the part of plaintiff's lawyers.

I believe two factors have combined to provide the safety the physician has enjoyed over the years: His

AGEMENT

knowledge of medicine, and his communication with patients.

Just as there is no substitute for an accurate diagnosis in medicine, there is no substitute for current medical knowledge when it comes to risk management. For most practitioners, this means frequent CME, and a well-developed sense of when to refer a patient to another physician. The state requires the former, and our profession encourages the latter.

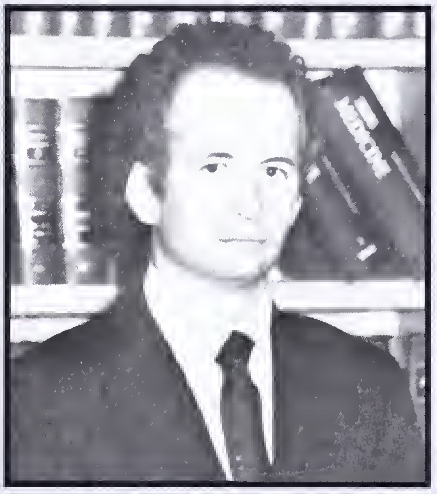
Things are not all so simple with regard to professional communication. There is no state law that mandates effective patient communication (though the requirement of "informed consent" comes close, in some areas). And we are not often called upon the carpet by our colleagues for having failed to develop such skills. Indeed, our intraprofessional courtesy generally results in silence when we learn of a colleague's misadventure in this area.

It is ironic that our ability to relate to patients is one which is seldom addressed in our formal medical education. Substantive medical knowledge is quite correctly emphasized; our manner of dealing with those whom we see, however, is seldom a matter of constructive scrutiny. This may be adequate for much of our education, but it is often totally inadequate for dealing with the real world of upset patients. Suits are brought by patients who are dissatisfied—even if the physician has committed no error. Satisfied patients do not bring lawsuits. This applies even if they hurt, are ill, or have lost function of some body system. How, then, does one go about the task of creating such satisfaction?

Clearly, there is no single answer, just as there is no single practice style. What works well for one physician, may be awkward and uncomfortable for the next. From the patient's perspective, however, there must be preception of at least one key point: the physician cares.

Patients go to doctors because they want to be cared for, and cared about. Often, we do neither. We may place them in hospitals for care, but the care will generally be provided by others, upon our order. We may see the patients only a scant few minutes a day. Similarly, we may treat their pathology quite capably, without more than a mild professional concern for the patient.

It is a sad fact that medical education courses produce physicians who confuse patient pathology for the patient. How many times has a discussion around an X-ray begun: "This is Mrs. Smith...."? It certainly is not Mrs. Smith. How many times has a consultation, say, a comatose patient ended with something like, "Thank you for referring this interesting patient"? The patient's pathology may be interesting, but it is difficult to understand how a personality devoid of cortical function would be thought "interesting". Patients generally do not appreciate such attitudes, and will quickly spot even their subtle expression.



For reasons quite removed from risk management, but certainly for risk management, we must communicate our concern to our patients. The patient who perceives a caring physician is far more likely to be satisfied with the care received, than is a patient who believes medicine is being dispensed by a technocrat. Most patients will tolerate mistakes, and they will tolerate misfortune, if they believe the doctor cares for them, and has done his or her honest best.

How a physician might choose to communicate his or her concern for the person of the patient is probably irrelevant. The importance is in the message, not the medium. My internist friend can't disguise that concern with words of grouchiness or gruffness. His patients see right through him. A surgeon I know sends a special little gift—flowers, or whatever—to each postop patient. To each his or her own. It is the genuineness of the communciation which counts.

An old adage holds that "the way to care for a patient, is to care for the patient." For effective risk management, we should communicate our understanding of that principal to the patient.

Lee S. Glass, M.D., J.D. — has addressed numerous groups on risk management including the international College of Surgeons and the Department of Orthopedics, University of Washington School of Medicine. Glass practices law with Faulkner, Banfield, Doogan & Holmes in Anchorage and Seattle. He is also obtaining postgraduate medical training at the University of Washington.

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"I'M GLAD I WENT INTO MEDICINE."

Born in Bellingham, Washington, Nancy Elliott went to the University of Washington. Beginning as a sociology major, in her junior year, she switched to pre-med and was graduated from the Woman's Medical College of Pennsylvania in 1954, the college's 100th anniversary.

The importance of schools for women prior to the women's liberation movement in the 60's, cannot be overestimated. Established to assist intelligent women in their goals for education and profession, they made possible generations of tutored women who otherwise would have languished in the oblivious roles society assigned by sex. This medical school is now known as the Medical College of Pennsylvania.

While Nancy Elliott had a rotating internship at Virginia Mason Hospital in Seattle, she says, unequivocally, that her best medical education was apprenticeship in which she learned in practice with the physicians in Anchorage in the mid 1950's. Claire Renn and Peter Koeniger (1) were her mentors in OB GYN; she scrubbed for Jack Sedwick (2) for seven years, learning much thereby.

Nancy was married to Harold Sydnam. Eventually, they were divorced. There are four children: a girl and three boys and one grandson.

In 1959, Milo Fritz, ophthalmologist and otolaryngologist, decided to demonstrated the feasibilities of establishing clinics in the Bush. He wrote various villages which were served by the Episcopal Church. Both Milo and Nancy are Episcopalians.

Since he felt that responsibilities were to be shared, he extracted the promise that the villages would: provide a place to stay; electricity; and personnel to man the 'recovery room', assist in surgery, and take the patients home. He felt that it was important to encouraged villagers to be active participants in the program. In return he, with the able assistance of his wife, Betsy, a nurse, would remove tonsils, pull teeth and examine ears and eyes.

The Alaska Chapter of the American Cancer Society sponsored Nancy Sydnam, for one-half her costs, to go and do pap smears. She ended up by doing pre-op physicals as well as general physicials when needed. Nancy diagnosed a child with heart disease whom the Episcopal Bishop flew to Tanana for evacuation to Anchorage. Having once had to use the Bishop's desk as an examining table, Nancy wryly remarks "It's hard to do physicals without a proper table."

Villages along the Yukon were the targets: Shageluk (patients came from Holikachuk), Huslia, Allakaket (patients came from Koyukuk and Hughes), Stevens Village, Beaver and Venetie. Nancy and Betsy travelled commercially; Milo flew his plane.

This was Nancy Syndnam's first contact with Alaskan Natives. She was overwhelmed with the incidence of alcoholism; sorrowed by the generation gap between old and young; astounded by the diet, especially water and flour pancakes. She marvelled at the summertime tents of the older generation with their woven willow floors. She remembers vividly blind Chief Henry who made snowshoes, having grooves along the sides to guide him in placing the holes.

The Sydnams owned a house on Sand Lake which had been built by a Pacific Northern Airlines (3) pilot and was insulated with corrugated paper and tin foil. They were to live there until Sid was transfered to Juneau in 1967. During the years in Anchorage, Nancy practiced with the Anchorage Medical and Surgical Clinic and in 1962 went into solo practice at 5th and Barrow.

Nancy Sydnam looks upon her years in Juneau as good years when she worked part time, as little as one half a day a week. She would occasionally do locum tenems for Henry Akiyama and Ken Moss, a pediatrician. However, she felt that she could function as a full time mother. She says that it is difficult to play the multiple roles of physician, wife and mother.

Having been involved in obstetrics for years and seen the marked need for perinatology which involved the baby both in utero and after birth, Nancy finally had time for a 6 month fellowship in perinatology at the University of Washington.

In 1972, the Sydnams returned to Anchorage. She started to practice half days with Claire Renn and in 1980, with children grown, started fulltime practice again.

Returning from Juneau was a new medical world. The old commraderie of a small medical community where everyone knew one another was gone. In the early years there had been a high doctor to patient ratio. She felt that while technology was lacking, the quality of care had been good.

She recalls being called to the Emergency Room to attend a man complaining of a severe headache that began suddenly one hour previously. When she looked into his mouth, she found a bullet hole with bullet embedded in the hard palate. The headache was diagnosed as a "Saturday Night Special." Cure was provided by neurosurgeon Perry

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(1) Peter Koeniger, in 1982, was practicing OB GYN in Hoquiam, WA.
(2) Jack Sedwick, a general surgeon, is retired in Bend, OR.
(3) PNA was absorbed by Western Airlines.

Mead.

In 1979, Nancy went to Kenya for three months to work at a Quaker Hospital. She felt that at best she could only suggest sterilization for the tribal women who were overburdened with children. She remembers one man who requested sterilization for his wife. His motive: a barren woman could be divorced.

She ponders on the roles of the woman physician. In her time, the non-aggressiveness of women was a disadvantage: at first she worked longer and harder for less; had a hard time seeking advantages for herself. This has changed. But she feels that the balancing act of family and practice for the woman physician has not become easier.

Nancy Sydman recently thoroughly enjoyed being co-surgeon in the microsurgery done to an Abyssinian cat from Eagle River. This was the first successful feline kidney transplant in North America. While the University of California at Davis had attempted it, they were not successful. They did send their protocol which the Alaska experience amended and enlarged. A hot line was kept open between Alaska and California during the surgery. The cat is doing well.

In 1984, Nancy and her sisters set up the Winnie Barclay Elliott Foundation in honor of their mother. Nancy and her sisters felt that they had an outstanding mother who in the accepted way of things received no recognition for the many things she had accomplished in life especially as mother and teacher. The Foundation honors persons for excellence in education. The first two recipients are Sister Jeanette LaRose and Flora MacDonald.

Nancy Sydnam believes that people should be trained to do things for themselves. It is a doctor's task to educate and medicate. She is vastly interested in what is being done in Norway, Sweden and Russia to train medical aids. She thinks that she would like to return to work in the Bush or perhaps in Nepal where there is much to do in immunization and childbirth.

Whatever she does, her life until now tells us that she will do it with dedication and skill.

Gwynneth Wilson
for the ASMA Auxiliary

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PRESIDENTS PAGE

The issues of Tort Reform and cost containment continue to occupy much of our attention this year. Happily there is much progress to report on the Tort Reform effort. House Bill 532, introduced by Representative Cotten, contains the issues thought most important by the Citizens' Coalition for Tort Reform. Senate Bill 377, sponsored by Senator Kelly, contains many, but not all of the same provisions. As all of you know, the introduction of a bill does not indicate that it or anything like it will be passed. We have a great deal to do. The Tort Reform assessment, which many of our members have gladly paid, has been critical to our efforts so far. For those members who have not paid the assessment, your support is critical and badly needed.

For those of you who may have heard the rumor that "Tort Reform is dead this year", do not be easily discouraged. Such an opinion is precisely what the opposition wants us to hold. On March 10th, we were able to meet at length with Governor Sheffield to discuss the issues of Tort Reform. He believes that something must be done about insurance problems generally. He, like all of our elected representatives, would like to have a "quick fix". Unfortunately no such quick fix is available. We must persist in our efforts to inform our fellow citizens and our elected representatives of the need for Tort Reform.

We have had excellent advise and support from many of our elected representatives. Others have not been as supportive. This Fall brings another election year. Right or wrong, the fact is that it takes money to get elected. Please remember to support those who will most effectively represent our views. Please contact David Johnson, ALPAC Chairman, for further information in that regard.

The issue of cost containment and the proper role of physicians in controlling costs is with us and will be a source of continuing concern for years to come. There is little doubt that the cost of medical care has escalated in recent years. Patients, employers, and government agencies are concerned about the present cost of medical care. Everyone is and should be concerned about the future costs and availability of medical care.

The Council of the Alaska State Medical Association, at its Winter meeting in Anchorage, voted unanimously to establish an ad hoc committee on cost containment. Laurie Bleicher, M.D. has accepted my request to serve as Chairman of the committee. She is a capable surgeon in Anchorage and brings a wealth of experience from other practice experiences to this task. She will need support and assistance from other members of our Association. Please volunteer to serve and support her in this difficult task.

As most of you know, both hospitals in Anchorage have in place a Preferred Provider Organization. The conditions of both PPO's are such that there exists significant disincentives to the patient to obtain medical care anywhere but with the institution in question. Obviously, this has significant long-term consequences to physicians and patients. Many questions arise. Is this the mechanism of health care funding that best serves the needs of the patient and physician? Apparently the institutions and their insurance contractors think so. Alaska doctors must know more about this entire problem.

Physicians receive approximately 20% of the Nation's health care dollar. Yet, they are said by many to control the expenditure of 80% of the health care dollar. There are, have been, and will be many proposed mechanisms to influence physician behavior in that regard. Mandatory second opinions prior to surgery represents one of these mechanisms. Is it effective? Are there other better methods? Are the facts as stated correct? Where does the money go, really? We hope the committee will be able to effectively address such questions.

Outpatient surgery has been demonstrated to be safe, effective, and more cost effective than inpatient care for many health problems. Such care in free-standing facilities is usually less expensive than in hospital-associated facilities. Hospital supporters argue that the provision of ICU, CCU and Neonatal Intensive Care units justify such disparities. Should that information be better documented or more generally available? Is it true? Is that the way most payers of health care cost want it to be? Should alternatives be sought? Again, these are questions we hope the Committee will address.

In his address to the Alaska State Medical Association Convention in Haines last year, Mr. Rick Ruben, then President of the Coalition for Health Care Cost Containment, made several observations. More information relative to the true costs of various components of the medical care system is held by the insurance companies than by any other sector. (Ours is an Information Age. He who has the information has the power. He who has the gold makes the rules.) Insurance companies, hospitals and other providers are, in general, more organized than physicians. They are, in general, better funded than most physician organizations. Right now, physicians enjoy significant influence over health care expenditures. Unless they use that influence in a more organized fashion, they will lose it. I had the opportunity to discuss these issues again with Rick recently. He hasn't changed his mind. As physicians we must become more aware of the impact of health care costs. We must understand the distribution of the health care dollar. We must work to implement positive changes to provide more value to the patient per dollar expended. Without information we cannot possibly know what should be done. Without organization we can't possibly do anything once we know what should be done. This Committee will be asking for your help and support. The information they gather will be important to us all.

David A. McGuire, M.D.
President, Alaska State Medical Association

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A Very Special Arts Festival For The Handicapped

1986 will be remembered as the year the medical community of Anchorage came to the rescue of The Rainbow Factory, Anchorage's version of the national Very Special Arts Festival for the Handicapped.

This five day event, held at the Anchorage Museum, provides workshops in music, drama, dancing, mime, sound, clowning, painting and sculpting, for over 1500 disabled, retarded and otherwise handicapped persons. The festival lost it's sponsor and was at the point of having to be cancelled when individual medical groups, hearing of the need, responded to meet the financial commitment. Five sponsors were located who were willing to underwrite one day each in order for the festival to continue.

Diagnostic Imaging of Alaska sponsored the first day, followed by the **Alaska Pediatric Association** on the second. The third and fourth days were underwritten by **Anchorage Orthopedic Association** and **Ophthalmic Associates.** The last day of the Rainbow Factory, which is open to the public, was sponsored by the **Anchorage Medical and Surgical Clinic.** This day alone was enjoyed by an appreciative crowd of approximately 1200.

The Anchorage Times, quoting Goodwin Trent of the Anchorage Arts Council, said "We almost didn't do it because of lack of funding, but the medical community this year saved the day for us. It's a group effort." A very special auxilian, Pat Wolf, is Director of Education at the Museum and feels this to be one of the most important and exciting events held there each year.

"When art speaks to a person, it doesn't care whether that person is in a wheelchair, whether he is sightless, or whether he can hear. Art doesn't discriminate" says Catherine Stadem of the Times. Hats are off to these sponsors who made the ninth Rainbow Factory a reality.

Gala Event For Scholarship Held In Fairbanks

The House of Wood, a popular art gallery in Fairbanks was the scene of a beautiful social event for the medical community. Funds were raised for the scholarship fund through a raffle on a beautiful print that was donated, through individual contributions, and through the sale of the Auxiliary print "Night Flight" by Auxilian Marianne Wieland.

Approximately $1,700 was realized by the effort which was coordinated by members Alice Lundquist, Janie McConkey and Mary Worrall. A memorial contribution was made in honor of Dr. Abe Cannon to provide a $500 scholarship to be awarded to a medical student.

According to Sally Brownsberger, state scholarship chairman, about 60 applications were received for the two $1,000 scholarships given by the State Auxiliary. The top 10 finalist's scholarship folders will be taken to the Convention at Mt. McKinley where the selection fo the two winners, plus the winner of the one time Abe Cannon Scholarship will be decided.

The Anchorage Medical Auxiliary donated $1,077 to the scholarship fund which was realized from interest earned on money left over from Pecabu car seat loaner program account.

Holiday Greeting Card

The first Holiday Greeting Card project to benefit AMA-ERF, was successful in raising $1,680 for medical schools. Kathie Wood, project chariman, expressed satisfaction that so many respondents were from areas outside Anchorage. A special feeling of goodwill was created among the medical families throughout the state to have greetings sent to all physician families at the holiday season by those listed on the card.

Children atending the State Medical Convention in June will have the opportunity to create drawings which will be featured on the 1987 card. A selection of six of these designs will by made by the AMA-ERF committee at Convention.

Special thanks to Auxilian Marcia Brown of Fairbanks for her delightful design on the 1985 Holiday Greeting Card.

Diptheria Immunization Projects

Auxilians in Anchorage were active with the massive diphtheria immunization project. The Red Cross has expressed their gratitude for the many volunteer hours spent by our members in locations throughout the city. Once again it was shown that our Auxiliary, working in coalition with other health organizations can create a strong union to meet the health needs of our various communities.

Carolyn Crouch

1986 ALASKA STATE MEDICAL CONVENTION

Mt. Mckinley Park Chalets
June 4-8, 1986

Meeting Schedule & Activities

Wednesday - June 4, 1986

7:30- 4:30	Regsistration & Ticket Purchase
8:30- 9:30	ASMA Council Meeting
10:00-12:00	ASMA Business Meeting
10:30-12:00	Exhibitor Set-Up
1:00- 3:30	Andrew Morely, M.D. "Physician Marketing"
3:45- 4:30	Larry Ford, M.D. Routine Screening in OB/TORCH Syndrome"
5:00- 6:30	Salmon Bake Dinner
6:30	Bus to Park Station Hotel
7:00- 8:00	Naturalist Program
8:00- 9:00	Libby Riddles—1985 Iditarod Winner
9:30-10:30	Visit the Exhibits Desserts & Nightcap Hosted by Searle Labs.

Thursday - June 5, 1986

8:00- 1:30	Registration & Ticket Purchase
8:00- 9:30	ASMA Business Meeting
9:30-10:00	Visit the Exhibits
10:00-10:45	Alain Enthoven, Ph.D. "What's Happening in U.S. Health Care Economy"
10:45-11:15	Larry Ford, M.D. "Diagnosis & Treatment: Serious Infections in OB/GYN"
11:30- 1:30	Box Lunch "Meet the Candidates" 11:30 Bob Richards 11:45 Joe Hayes 12:00 Ronald Somerville 12:15 Bob McGrane 12:30 Steve Cowper 12:45 Joe Vogler
1:30	Bus for Early River Float Trip or/Hike Up Ridge
4:30	Transport to Bar-B-Q
5:00	Bar-B-Q Dinner Games Healy Express or Canyon-Run Raft Trip

Friday - June 6, 1986

8:00-10:00	AAFP Fun Run
10:00-10:45	Steven DeVore "Methods of Success: The New Psychology of Achievement"
10:45-11:30	Alain Enthoven, Ph.D. "Cost Effectiveness in Medical Care"
12:00- 2:00	Specialty Luches: **College of Surgeons** -Chalet Bar **Pediatrucians**-Dining Room **Orthopedics**-Executive Suite
2:00- 4:00	ASMA Business Meeting
4:15- 5:15	Walter Trudeau, M.D. "Gastrointestinal Stasis Syndrome"
6:30 Group Dinner	Speakers: Nancy Hogshead, Arliss Sturgulewski, Dick Randolph

Saturday - June 7, 1986

7:30- 4:30	Registration
8:00-10:00	ASMA Business Meeting
10:15-10:30	Richard Garner, M.D. "Use of Bone Biopsy"
10:30-11:15	Walter Trudeau, M.D. "Reflux Esophagitis"
10:30	Exhibitors dismantle exhibits
12:00- 2:00	Auxiliary Lunch & Annual meeting ALPAC Lunch Meeting Poltical Speaker
2:00- 2:45	O.W. Frost, Ph.D. "Georg Steller - Alaska's First Physician"
2:45- 3:30	Jay Ofsthun, CPA - Bigler, Kohler & Obendorf "1986 Tax Laws"
3:30- 5:00	ASMA Business Meeting
6:00	No Host cocktails
7:00	ASMA President's Banquet General Chuck Yaeger - Special Speaker Award Presentations, and Drawings

Sunday - June 8, 1986

9:00-11:00	ASMA Council Meeting

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John Middaugh, M.D.
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__ References are cited in the text by numerals enclosed in parentheses. The reference section is typed double-spaced on sheets separate from the text and is numbered consecutively in the order in which references are cited in the text. Included are last names and initials of all authors, title of article, name of publication, volume, inclusive pages and year published. Abbreviations will conform to those used in **Index Medicus**.

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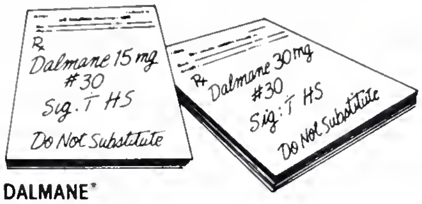
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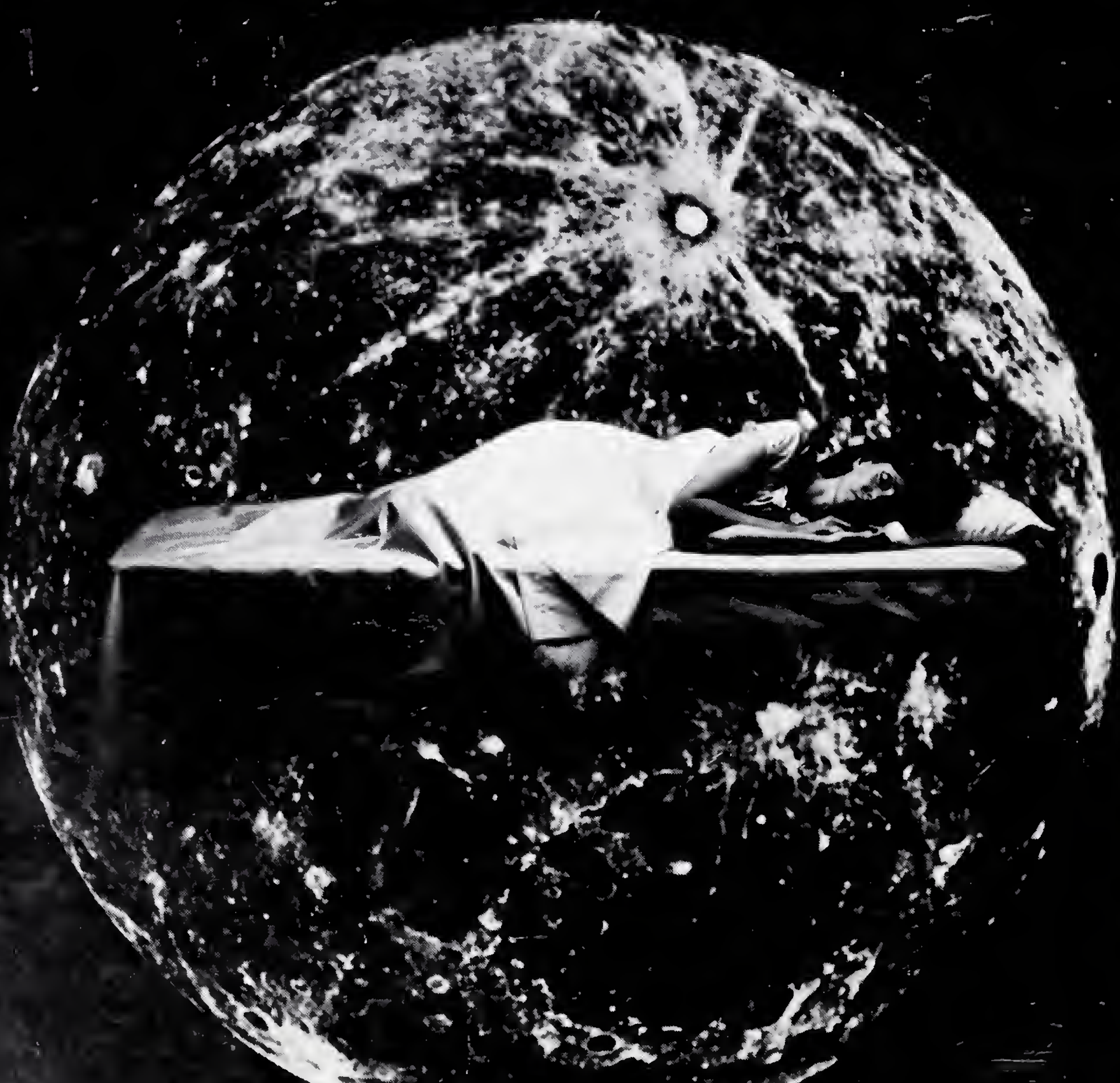

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ALASKA MEDICINE

Volume 28, Number 3
July/August/September

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ASSETS	$118,505,000	$101,620,000	$91,691,000	+29.24
POLICYHOLDERS' SURPLUS	$ 31,773,000	$ 27,820,000	$26,009,000	+22.16
GROSS WRITTEN PREMIUM	$ 24,971,000	$20,703,000	$17,829,000	+40.06
NET INVESTMENT INCOME	$ 7,346,000	$ 6,509,000	$ 6,094,000	+20.54
RATIO OF NET WRITTEN PREMIUM TO SURPLUS	.79:1	.59:1	.45:1	—
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ALASKA MEDICINE

Official Journal of the Alaska State Medical Association

2401 E. 42nd Avenue, Anchorage, Alaska 99508

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7th International Congress on Circumploar Health
June 8—12, 1987, Umea, Sweden

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GENERAL INFORMATION

BACKGROUND. The first six symposia on circumpolar health were held in F banks, Alaska (1967), Oulu, Finland (1971), Yellowknife, C.N.W.T. (1974), Novo birsk USSR (1978), Copenhagen, Denmark (1981) and Anchorage, Alas (1984). The reasons for holding a symposium every three years are: 1) to bri together medical scientists, health care delivery specialists, health administ tors and health consumers to discuss the state of the art in their respective fiel 2) to allow national and international participants to observe and discuss t health situation in their own countries, and 3) to relate solutions to health pro lems in other parts of the world to the unique problems of the circumpo regions.

During the Congress the International Union for Circumpolar Health will have first general assembly.
PRESENTATIONS will include invited speakers, free papers and poster sessio The scientific sessions will be scheduled over four days. Official language of t congress will be English. An exhibition of scientific and technical interest a post-congress meetings will be arranged. A post-congress Scandinavian Virolo Meeting in Meeting in Umea, June 12-14, 1987, is planned.

Main topics of the Congress:

1. Occupation and health in circumpolar areas
2. Environment in the Arctic and Subarctic. Ecological, social, behavioral, p chological, geographic, and economic aspects in relation to health (includi problems in small groups, isolation and self-determination of indigeno peoples, acculturation problems)
3. Health care planning and delivery, vital statistics
4. Health care education
5. Health care engineering (including environmental control and sanitation)
6. Industrial development and health (including industrial hygiene hazards co cerning illumination, ventilation and use of modern technology in circump lar regions)
7. Cold injury, hypothermia; adaptation, physiology and pathology in cold c mates
8. Genetics and epidemiology
9. Anthropology and demography
10. Oral health
11. Nutrition
12. Diseases of the eye, ear, nose and throat
13. Infectious diseases
14. Cancer and other (chronic) diseases
15. Mental health, alcoholism, drug and child abuse
16. Emergency medical services and communication (including injury preve tion)
17. Biological and circadian rhythms
18. Arctic medical history and traditional medicine
19. Maternal and child health

Social Program: Reception, congress banquet, concert, sightseeing, excursio visit to museum and handicraft exhibition. Shopping tours a a boat trip to the City of Vasa in Finland will be arranged. Pre-and post-congress tours to Northern Scandinavia, No Cape, Lapland and the Soviet Union are planned.

Deadline for abstracts: January 15, 1987.

COST SAVINGS OF REGIONALIZED PERINATAL CARE FOR LOW BIRTHWEIGHT INFANTS:

PART II. COST OF HOSPITALIZATION AND MORBIDITY

Jack Jacob, M.D.[1]

Introduction

In Part I of this series, we discussed the role of maternal transport and referral in improving the survival of LBW infants in Alaska. Part II deals with the effects of maternal transfer and referral on the cost of hospitalization and long-term morbidity.

Methods

The study was conducted on all low birthweight infants (birthweight less than 2500 gm) who were cared for at the Newborn Intensive Care Unit at Providence Hospital from 1980 through 1984. Charts of all infants were reviewed retrospectively. Data extracted for the present study consisted of the following: birthweight, race, outcome, place of birth, neonatal vs. maternal transfer, length of tertiary care hospital stay, 1 and 5 minute Apgar scores, presence or absence of intraventricular hemorrhage, length of time on mechanical ventilator, presence of severe chronic lung disease and severe neurological disability. In addition, the average daily cost of ventilated and non-ventilated infants was obtained from random selection of 21 ventilated and non-ventilated infants in 1985 that were hospitalized at the Newborn Intensive Care Unit at Providence Hospital. The severity of intraventricular hemorrhage was graded on the system of Papille et al. (1).

Results

Figure 1 shows length of tertiary care hospitalization for the combined years 1982 through 1984 for inborn and outborn LBW infants. For infants $<$ 1500 gm birthweight, the average length of hospital stay was 121 days for outborn infants vs. 53 days for those inborn. For infants between 1500 and 2499 gm, the average length of hospitalization was 25 days for infants outborn vs. 10 days for those that were inborn.

On rare occasions an infant may require very prolonged hospitalization because of severe chronic disability. Data on these infants for the years 1982-1984 that were hospitalized over one year are shown in Table I. Some of these infants were discharged but readmitted soon thereafter for periods over one year. The incidence of infants requiring over one year of hospitalization was 15-fold greater for outborn infants. The fact that such chronic disability is not solely a problem of the tiny LBW baby is demonstrated by the fact that 4 of the 6 such outborn infants weighed over 1500 gm at birth.

Figure 2 shows the incidence of low Apgar scores at 1 and 5 minutes by place of birth for the years 1982 through 1984. Low Apgar scores were defined as 1 or 5 minute Apgar scores less than 6. For infants less than 1500 gm, the incidence of low Apgar scores at 1 minute for inborn infants was 71% vs. 66% for outborn infants. By 5 minutes of age, the incidence of low Apgar scores decreased to 26% in inborn infants vs. 40% in outborn infants. For infants between 1500 and 2499 gm, low 1 minute Apgar scores were obtained for 38% of inborn infants vs. 50% for outborn infants. At five minutes of age, this decreased to 6.5% for inborn infants vs. 20% for outborn infants. The results document more effective resuscitation at the tertiary care center.

---

[1] Clinical Director, Alaska Newborn Care Project, 3300 Providence Drive, Suite 05, Anchorage, Alaska 99508.

Figure 3 shows the time spent on mechanical ventilation for inborn and outborn infants for the years 1982 through 1984, combined. In order to prevent bias in the data, infants dying at less than 14 days of age were excluded. For infants < 1500 gm, the average duration of mechanical ventilation was 19 days in inborn infants vs. 25 days for outborn infants. Similarly, for infants weighing between 1500 and 2499 gm, the average duration of mechanical ventilation was 3.8 days for inborn infants vs. 8.6 for outborn infants.

Figure 4 shows the incidence of intraventricular hemorrhage in inborn vs. outborn infants for the years 1982 through 1984, combined. For infants < 1500 gm, all grades of intraventricular hemorrhage occurred in 56% of inborn infants vs. 74% of outborn infants. For infants < 1500 gm, severe grade 3-4 hemorrhages occurred in 19% of inborn infants vs. 36% of outborn infants. For babies between 1500 and 2499 gm, the incidence of all grades of intraventricular hemorrhage was 13% for inborns vs. 24% for outborns. For severe grade 3-4 hemorrhages, the incidence of intraventricular hemorrhage for the infants between 1500 and 2499 gm was 0.1% for inborn infants vs. 8.7% for outborn infants.

These results not only indicate a lower incidence of intraventricular hemorrhage in all inborn infants, they also demonstrate that a greater percentage of severe hemorrhages occurred in outborn infants.

Although neurological assessment prior to discharge cannot predict long-term neurological outcome, infants that have severe disability may be diagnosed at discharge. Data on such infants for the years 1982-1984 are shown in Table I. The incidence of severe neurological disability was four-fold higher in outborn infants. The fact that such disability is not solely a problem of the tiny LBW baby is demonstrated by the fact that 7 of the 8 such outborn infants were greater than 1500 grams.

Another significant contributor to morbidity is chronic lung disease that results from oxygen and ventilator therapy. Such infants require prolonged hospitalization, have poor growth, may have developmental delay, and may require frequent re-hospitalization for pneumonia and congestive heart failure. Data on such infants that required prolonged hospitalization and/or home oxygen therapy for the years 1982-1984 are also shown in Table I. The incidence of severe chronic lung disease was three-fold higher in outborn infants. Once again, a majority (5/8) of the outborn infants were > 1500 gm.

Discussion

The implication of this study is that tremendous cost savings can be appreciated from regionalization of perinatal care if an emphasis is placed on the delivery of all LBW babies at the tertiary care center. The cost in terms of lives saved was discussed in Part I (2).

The cost of caring for a LBW infant at Providence Hospital averaged $1,414 per day in 1985. The average cost of an infant that needed mechanical ventilation was $1,649 per day compared to $1,132 per day for an infant not needing ventilation. Using these figures, combined with the data on length of tertiary hospital stay in Figure 1, cost saving can be calculated. If all LBW infants that were outborn in 1984 were born at a tertiary care center, a total of 1,024 patient days would be saved. At an average cost of $1,414 per day, one could appreciate a savings of $1,447,936 per year for tertiary care hospitalization. Since many of these patients were transferred to Level II hospitals for the remainder of their hospitalization, and since physician services are not included in these figures, they underestimate the total cost of hospitalization. Furthermore, with such an emphasis, extremely prolonged hospitalization would become a rare event. This would alleviate a tremendous financial and emotional burden on families and third-party payors.

It is extremely difficult to estimate the actual cost savings based on the decreased morbidity of inborn infants. Both Apgar scores and intraventricular hemorrhage are two perinatal events that might affect long term neurological morbidity. The long term effect of improvement in 5 minute Apgar scores is difficult to document. Grade 3-4 intraventricular hemorrhage, however, has been shown to result in hydrocephalus requiring shunting, seizures, and significant developmental delay (3-5). Using data from Figure 4 and 1984 admission data, 26 less LBW infants with Grades 3-4 IVH would have resulted if all such infants were inborn. Assuming a yearly cost of $20,000 per year in rehabilitation services, special education, subsequent hospitalization costs, etc., would result in additional yearly cost savings of $520,000.

This study demonstrates the significant morbidity that is associated with the delivery of a LBW infant out of the tertiary care setting. Similar results have been obtained elsewhere (6-9), including a lower incidence of intraventricular hemorrhage for maternal transports (10). Surprisingly, the results show that a significant portion of the morbidity was associated with the larger LBW baby (birthweight 1500-2499 gm). Similar studies done elsewhere have generally shown that the greatest benefit of maternal transport occurs in infants weighing less than 1500 gm at birth (11). There are two possible reasons for the differences in Alaska. First, VLBW infants born out of the tertiary care setting generally do not survive long enough to develop chronic problems. Secondly, many rural Alaskan hospitals have extremely limited capabilities for effective obstetric and neonatal management of any LBW infant. This is primarily related to the small number of deliveries that are done in such institutions. Despite motivated personnel and expenditure of resources, expertise is difficult to maintain for events that occur infrequently.

The implications of this study are clear. Physicians practicing obstetrics are the only ones that can substantially alter the morbidity and mortality associated with the birth of a low birthweight infant. The study, however, also has implications for third-party payors, state planning agencies, and malpractice insurers, since they also have the ability to change medical practice by controlling monetary factors involved in the delivery of health care.

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Figure 1

Length of Tertiary Care Hospitalization for 1982 - 1984 According to Location of Birth

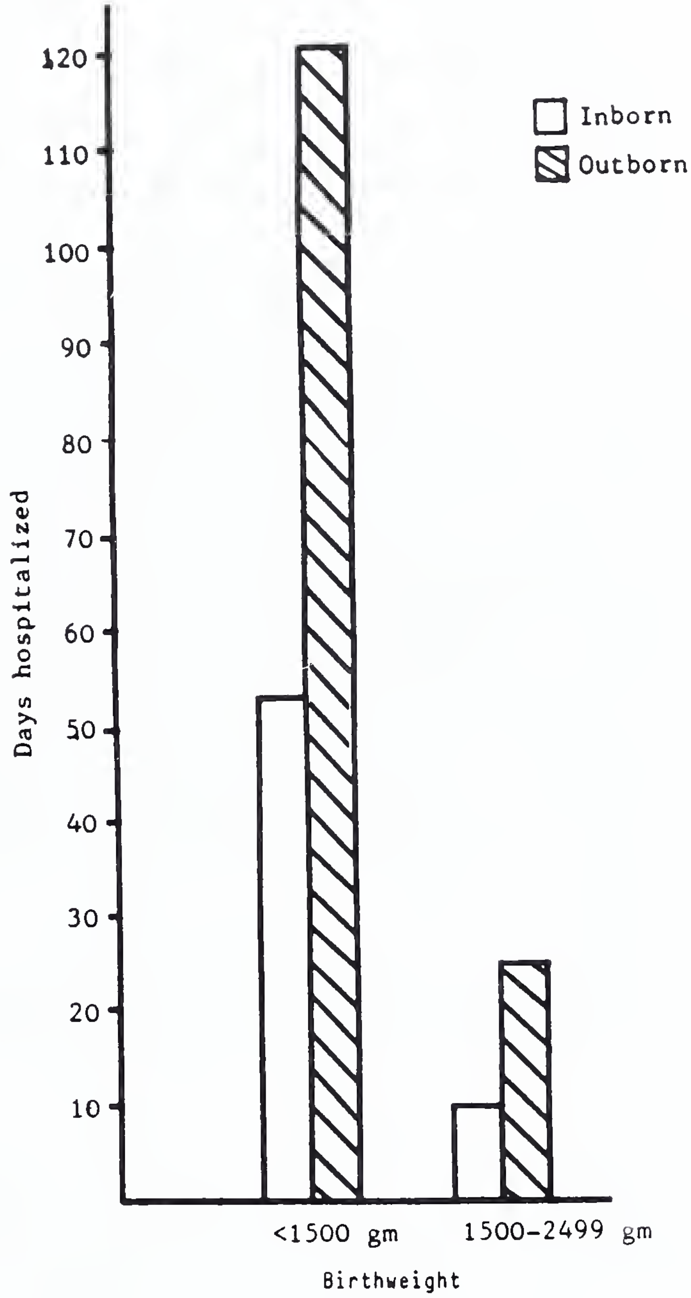
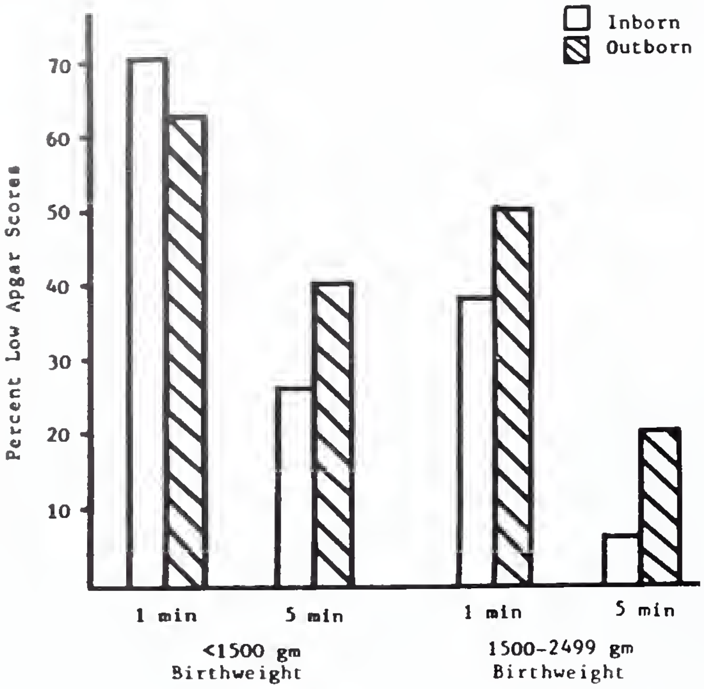


Table I
Chronic Hospitalization and Disability: 1982-1984

	Outborn N = 168	Inborn N = 408
Hospitalized over 1 year	6 (3.6%)	1 (0.2%)
Severe neurological disability at discharge	8 (4.8%)	5 (1.2%)
Chronic lung disease	8 (4.8%)	6 (1.5%)

Figure 2

Incidence of Low Apgar Scores* at 1 and 5 minutes by place of birth: 1982-1984



*Apgar score <6

Figure 4

Incidence of Intraventricular Hemorrhage (IVH): 1982 - 1984

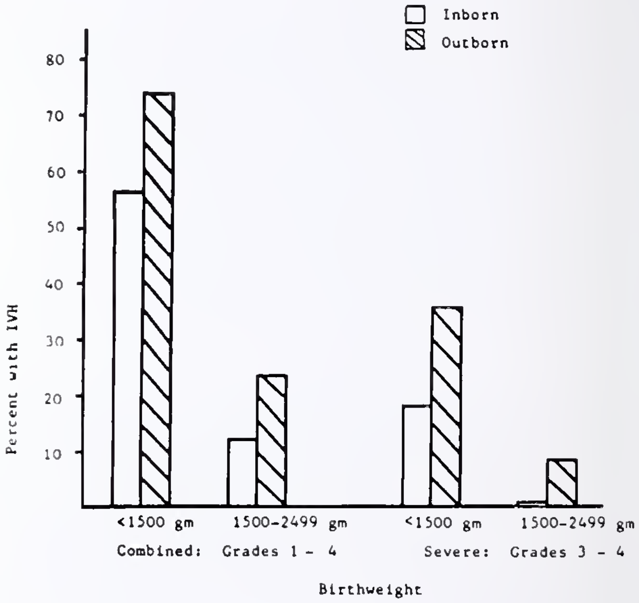
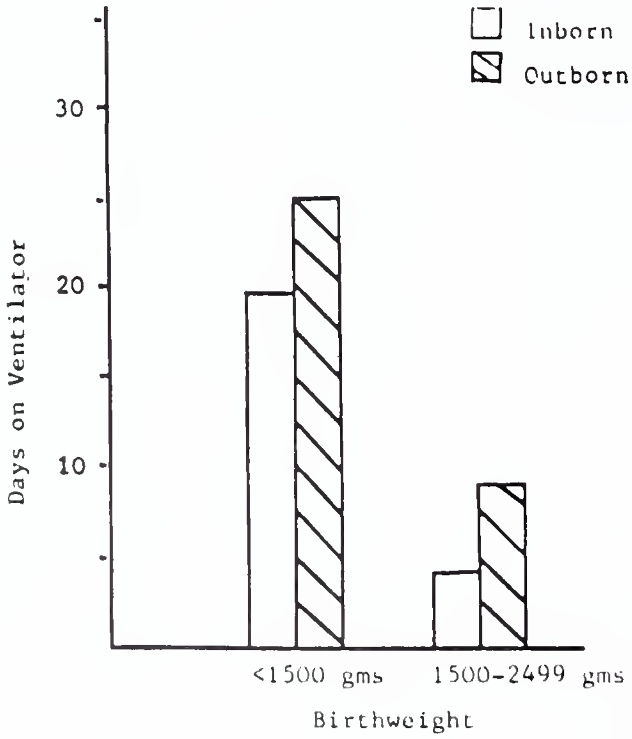


Figure 3

Length on Mechanical Ventilator*: 1982 - 1984



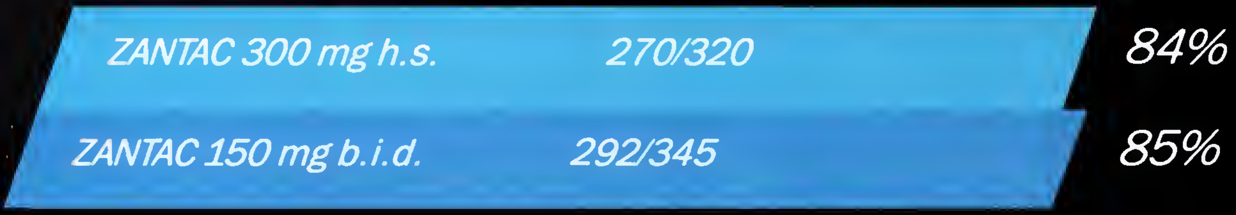
* Excludes deaths <14 days of age

CONTROL
ACID RAIN
with once-a-night
h.s. therapy for active
duodenal ulcers

Now, one tablet at bedtime

Controls nocturnal acid to relieve pain and heal duodenal ulcers

Heals active duodenal ulcers after 4 weeks in most patients*[1]



*In well-controlled, double-blind, multicenter trials, ZANTAC 300 mg h.s. healed active duodenal ulcers in 84% of patients after 4 weeks. After 8 weeks, healing rates may be higher with ZANTAC 150 mg b.i.d. (92%) than with ZANTAC 300 mg h.s. (87%).

Relieves pain and other symptoms as effectively as ZANTAC 150 mg b.i.d.[1]

Zantac® 300

ranitidine HCl/Glaxo 300 mg tablets

Once-daily dosing may enhance compliance in patients for whom dosing convenience is important

Side-effects profile comparable to ZANTAC 150 mg b.i.d.[1-3]

Headache—sometimes severe—has been reported. Rare effects on the CNS, cardiovascular, GI, hepatic, and integumental systems have been observed, as well as rare cases of hypersensitivity reactions. See ADVERSE REACTIONS section of Brief Summary of Product Information before prescribing.

No significant interference with the hepatic cytochrome P-450 enzyme system at recommended doses

ZANTAC 300 mg h.s. had no significant drug interactions with theophylline or warfarin. The bioavailability of certain medications whose absorption is dependent on a low gastric pH may be altered when ZANTAC or other medications which decrease gastric acidity are dministered.

Glaxo/ROCHE

See next page for references and Brief Summary of Product Information.

It is not known exactly how much acid inhibition s needed to heal ulcers.

IN ACTIVE DUODENAL ULCERS

Once-a-night h.s. therapy controls acid rain

Zantac® 300
ranitidine HCl/Glaxo 300 mg tablets

Now...two effective regimens to treat active duodenal ulcers

References: 1. Data available on request, Glaxo Inc. **2.** Ireland A, Colin-Jones DG, Gear P, et al: Ranitidine 150 mg twice daily vs 300 mg nightly in treatment of duodenal ulcers. *Lancet* 1984;2:274-275. **3.** Colin-Jones DG, Ireland A, Gear P, et al: Reducing overnight secretion of acid to heal duodenal ulcers. *Am J Med* 1984; 77 (suppl 5B):116-122.

ZANTAC® 150 Tablets
(ranitidine hydrochloride)
ZANTAC® 300 Tablets
(ranitidine hydrochloride)

BRIEF SUMMARY OF PRODUCT INFORMATION

INDICATIONS AND USAGE: ZANTAC® is indicated in:
1. Short-term treatment of active duodenal ulcer. Most patients heal within four weeks. Studies available to date have not assessed the safety of ranitidine in uncomplicated duodenal ulcer for periods of more than eight weeks.
2. Maintenance therapy for duodenal ulcer patients at reduced dosage after healing of acute ulcers. No placebo-controlled comparative studies have been carried out for periods of longer than one year.
3. The treatment of pathological hypersecretory conditions (eg, Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of active, benign gastric ulcer. Most patients heal within six weeks and the usefulness of further treatment has not been demonstrated. Studies available to date have not assessed the safety of ranitidine in uncomplicated, benign gastric ulcer for periods of more than six weeks.
5. Treatment of gastroesophageal reflux disease. Symptomatic relief commonly occurs within one or two weeks after starting therapy. Therapy for longer than six weeks has not been studied.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain.

CONTRAINDICATIONS: ZANTAC® is contraindicated for patients known to have hypersensitivity to the drug.

PRECAUTIONS: General: 1. Symptomatic response to ZANTAC® therapy does not preclude the presence of gastric malignancy.
2. Since ZANTAC is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function (see **DOSAGE AND ADMINISTRATION**). Caution should be observed in patients with hepatic dysfunction since ZANTAC is metabolized in the liver.

Laboratory Tests: False-positive tests for urine protein with Multistix® may occur during ZANTAC therapy, and therefore testing with sulfosalicylic acid is recommended.

Drug Interactions: Although ZANTAC has been reported to bind weakly to cytochrome P-450 in vitro, recommended doses of the drug do not inhibit the action of the cytochrome P-450-linked oxygenase enzymes in the liver. However, there have been isolated reports of drug interactions which suggest that ZANTAC may affect the bioavailability of certain drugs by some mechanism as yet unidentified (eg, a pH-dependent effect on absorption or a change in volume of distribution).

Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no indication of tumorigenic or carcinogenic effects in lifespan studies in mice and rats at doses up to 2,000 mg/kg/day.

Ranitidine was not mutagenic in standard bacterial tests (*Salmonella, E coli*) for mutagenicity at concentrations up to the maximum recommended for these assays.

In a dominant lethal assay, a single oral dose of 1,000 mg/kg to male rats was without effect on the outcome of two matings per week for the next nine weeks.

Pregnancy: ***Teratogenic Effects: Pregnancy Category B:*** Reproduction studies have been performed in rats and rabbits at doses up to 160 times the human dose and have revealed no evidence of impaired fertility or harm to the fetus due to ZANTAC. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: ZANTAC is secreted in human milk. Caution should be exercised when ZANTAC is administered to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Use in Elderly Patients: Ulcer healing rates in elderly patients (65 to 82 years of age) were no different from those in younger age groups. The incidence rates for adverse events and laboratory abnormalities were also not different from those seen in other age groups.

ADVERSE REACTIONS: The following have been reported as events in clinical trials or in the routine management of patients treated with oral ZANTAC®. The relationship to ZANTAC therapy has been unclear in many cases. Headache, sometimes severe, seems to be related to ZANTAC administration.

Central Nervous System: Rarely, malaise, dizziness, somnolence, insomnia, and vertigo. Rare cases of reversible mental confusion, agitation, depression, and hallucinations have been reported, predominantly in severely ill elderly patients.

Cardiovascular: Rare reports of tachycardia, bradycardia, and premature ventricular beats.

Gastrointestinal: Constipation, diarrhea, nausea/vomiting, and abdominal discomfort/pain.

Hepatic: In normal volunteers, SGPT values were increased to at least twice the pretreatment levels in 6 of 12 subjects receiving 100 mg qid IV for seven days, and in 4 of 24 subjects receiving 50 mg qid IV for five days. With oral administration there have been occasional reports of reversible hepatitis, hepatocellular or hepatocanalicular or mixed, with or without jaundice.

Musculoskeletal: Rare reports of arthralgias.

Hematologic: Rare reports of reversible leukopenia, granulocytopenia, thrombocytopenia, and pancytopenia.

Endocrine: Controlled studies in animals and man have shown no stimulation of any pituitary hormone by ZANTAC and no antiandrogenic activity, and cimetidine-induced gynecomastia and impotence in hypersecretory patients have resolved when ZANTAC has been substituted. However, occasional cases of gynecomastia, impotence, and loss of libido have been reported in male patients receiving ZANTAC, but the incidence did not differ from that in the general population.

Integumental: Rash, including rare cases suggestive of mild erythema multiforme, and, rarely, alopecia.

Other: Rare cases of hypersensitivity reactions (eg, bronchospasm, fever, rash, eosinophilia) and small increases in serum creatinine.

OVERDOSAGE: There is no experience to date with deliberate overdosage. The usual measures to remove unabsorbed material from the gastrointestinal tract, clinical monitoring, and supportive therapy should be employed.

Studies in dogs receiving doses of ZANTAC® in excess of 225 mg/kg/day have shown muscular tremors, vomiting, and rapid respiration. Single oral doses of 1,000 mg/kg in mice and rats were not lethal. Intravenous LD_{50} values in rat and mouse were 83 and 77 mg/kg, respectively.

DOSAGE AND ADMINISTRATION: Dosage Adjustment for Patients with Impaired Renal Function: On the basis of experience with a group of subjects with severely impaired renal function treated with ZANTAC®, the recommended dosage in patients with a creatinine clearance less than 50 ml/min is 150 mg every 24 hours. Should the patient's condition require, the frequency of dosing may be increased to every 12 hours or even further with caution. Hemodialysis reduces the level of circulating ranitidine. Ideally, the dosage schedule should be adjusted so that the timing of a scheduled dose coincides with the end of hemodialysis.

HOW SUPPLIED: ZANTAC® 150 Tablets (ranitidine hydrochloride equivalent to 150 mg of ranitidine) are white tablets embossed with "ZANTAC 150" on one side and "Glaxo" on the other. They are available in bottles of 60 tablets (NDC 0173-0344-42) and unit dose packs of 100 tablets (NDC 0173-0344-47).

ZANTAC® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shaped tablets embossed with "ZANTAC 300" on one side and "Glaxo" on the other. They are available in bottles of 30 (NDC 0173-0393-40) and unit dose packs of 100 tablets (NDC 0173-0393-47).

Store between 15° and 30°C (59° and 86°F) in a dry place. Protect from light. Replace cap securely after each opening.

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DEATH FROM CANCER IN ALASKA

Anne P. Lanier, M.D., M.P.H.[1]

Abstract

Data on cancer deaths in Alaska are presented for the decade 1970-1979. Approximately 220 persons died from cancer each year. The most frequent type of cancer causing death was lung cancer. Compared with rates for the United States, Alaskan cancer death rates overall were low except for nonwhite women. Despite generally low overall death rates, excess deaths occurred in Alaskan white women for cancers of the lung and esophagus, in nonwhite men for cancers of the nasopharynx and liver and biliary tract, and in nonwhite women for salivary gland, large intestine, and kidney cancer.

Introduction

The report of the cancer research center feasibility study (1) emphasized the lack of data available on the occurrence of cancer in the State of Alaska. Although cancer incidence data are available for Alaskan Natives and were summarized in a previous article (2), no data for nonNatives have been available since the mid-1970s with the demise of the Washington/Alaska Automated Tumor Registry and its short-lived offspring, the Alaska State Tumor Registry. For Alaska, **estimated** deaths and new cases are included each year in the January/February issue of "CA - a Journal for Clinicians". Only the total number of cancers and for nine major sites are listed. There are no estimates by sex, age, and race, although marked differences occur in cancer patterns in Alaskan population groups.

Until the fairly recent proliferation of population-based cancer registries in the United States, death certificate data were the only source of information on cancer occurrence and trends. Limited information on cancer deaths in Alaska is made available periodically in reports from the Alaska Department of Health and Social Services (3).

Recently cancer mortality data for the period 1950-1979 for each state were compiled by the National Cancer Institute and Environmental Protection Agency (4). The purpose of this report is to make these data available to Alaskans.

Materials

The original publication includes three volumes of tables of cancer death rates for each organ site and all sites combined by sex, race (white vs. nonwhite), and state (4). Rates for each state have been adjusted (i.e., corrected for differences in age distribution of the populations) and can, therefore, be compared with each other or with the overall U.S. rates. Differences in the number of deaths recorded from those expected were tested for statistical significance. Rates are based on submission of death certificate information from the states to the National Center for Health Statistics. Data are given for only two categories, white and nonwhite. However, since Native Alaskans (Eskimos, Indians, Aleuts) comprised 80% of the nonwhite population in 1970 and 70% in 1980, the nonwhite death rates in Alaska largely reflect those of the Natives. Rates are summarized generally by state for each of three decades (1950-59, 1960-69, and 1970-79) except for Alaska for which data are presented only for the last two decades.

Results

Tables 1 and 2 present the numbers and rates of cancer deaths in Alaska compared with rates for the entire United States. Only rates for the 1970-79 decade are included in this article because they are the most recent, probably the most accurate, and in most instances do not differ from those of 1960-69.

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In Alaska, an average of 165 cancer deaths occurred annually in whites and 56 in nonwhites in the decade 1970-79. Lung cancer was the most frequent cause of cancer death in both whites and nonwhites. Other leading cancers in whites were large intestine/rectum, breast, leukemia/lymphoma, and brain/nervous system. In nonwhites, the most frequently reported cancer deaths following lung were large intestine/rectum, liver/biliary, leukemia/lymphoma, and stomach. These data can be compared with the most frequently reported causes of cancer death in the United States in men and women combined: lung, large intestine/rectum, breast and leukemia/lymphoma.

Age-adjusted cancer mortality rates for Alaskan white and nonwhite men and women in 1970-79 are compared with those for the United States overall. For all cancers combined, rates are somewhat lower in Alaska than in the United States, except in nonwhite women, but these differences are not statistically significant. For specific cancer sites, significant excess deaths occurred in nonwhites of both sexes for cancers of the nasopharynx, liver and biliary tract, and those with site unspecified. In nonwhite women, excess deaths from cancers of the salivary gland, large intestine, and kidney were also reported. Nearly identical patterns were found for cancer deaths in nonwhites in the decade 1960-69, except that excess deaths also occurred in women from cancer of the endocrine glands and in both men and women from kidney cancer. In Alaskan whites, significantly increased cancer deaths were reported only in women and for only two sites: lung and esophagus. Esophageal cancer deaths were also increased in white women in the 1960-69 period, whereas deaths from lung cancer were not. Excess cancer deaths were not identified for white Alaskan males in either period.

Discussion

The excess cancer deaths in nonwhites largely reflect the incidence patterns among Alaskan Natives which have been well documented (2). However, excess deaths in nonwhite women from cancer of the large intestine were not expected since a cancer survey in the mid-1970's (5) did not document an increased incidence. This finding may indicate relatively poor survival for patients with this cancer. Because excess deaths were also identified for cancers with primary site unspecified, it is possible that excess deaths also occurred for cancers of a few other sites but were not recognized because they are not assigned to specific sites. As noted, deaths for cancers of the lung and esophagus were in excess of expected for white women but not men.

Questions are always raised about the accuracy of death certificate information. With regard to cancer deaths in Alaskan residents, there is concern about potential underreporting of those who are treated and die outside of Alaska or who actually move out of state because of a terminal illness. Deaths are notified to the State which the patient lists as his usual residence, not the location of death, which would resolve part of the problem. Although cancer death rates for both groups of Alaskan men are lower than the overall U.S. rates, those for Alaskan women (white and nonwhite) equal or exceed those for the United States as a whole. Rates for Alaska that differed significantly from those for the rest of the United States showed an excess, not a deficit. Finding such an excess despite concern for underreporting make the findings even more remarkable. Finding excess rates could also reflect an error in estimation of the population, a particular problem in a rapidly growing state. However, under-enumeration of the population would be expected to result in excess rates for all cancer sites, not just a select few.

Conclusion

Data for nonwhites confirm the previously documented differences in cancer patterns in Alaskan Natives. Whereas few differences were noted in the number of cancer deaths for whites in Alaska compared with the United States, excesses were found in women for two cancer sites: lung and esophagus. Although these patterns may reflect the increased prevalence of smoking in the Alaskan population (7), if these patterns persist in the data for the 1980's, further investigation is warranted.

In Alaska, as elsewhere, the leading cause of death from cancer is lung cancer—which is preventable. The next leading causes are colorectal and breast cancer—sites for which screening and early detection should result in decreased mortality. An active program of vaccination for hepatitis B and AFP screening for hepatitis B surface antigen carriers in Alaskan Natives should reduce the high mortality from liver cancer (6). Although additional information would be useful, existing data are sufficient to document that in Alaska, the greatest decline in cancer mortality can be achieved through "stop smoking" programs.

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Table 1
Cancer Mortality in Alaskan White

	Males			Females		
Cancer Site	Alaska Deaths	Alaska Rate	U.S. Rate	Alaska Deaths	Alaska Rate	U.S. Rate
All Combined	941	184.4	204.1	710	130.9	131.7
Lip	1	.5	.2	—	—	.0
Oral/Tongue	17	3.3	4.6	6	1.1	1.5
Salivary Gland	2	.3	.4	—	—	.2
Nasopharynx	6	1.0	.4	1	.0	.2
Esophagus	23	4.5	4.4	11	2.8*	1.2
Stomach	37	9.8	9.0	16	4.7	4.3
Large Intestine	74	13.5	20.0	54	11.3	16.4
Rectum	18	4.2	5.8	16	4.4	3.4
Liver/Biliary	26	4.3	4.6	15	3.5	3.8
Pancreas	45	8.6	10.9	22	4.1	6.7
Nose/Nasal/Sinus	2	.5	.4	2	.4	.2
Larynx	8	2.5	2.7	1	.1	.4
Trachea/Lung	341	64.5	64.0	137	24.1*	15.3
Bone	6	.9	1.0	4	.3	.6
Conn/Soft Tissue	6	.8	1.1	10	1.0	.8
Melanoma	7	1.0	2.4	4	.3	1.5
Other Skin	3	.6	1.0	1	.3	.4
Breast	1	.1	.3	154	26.0	27.0
Uterus	—	—	—	18	3.7	4.3
Cervix	—	—	—	20	3.3	4.2
Ovary	—	—	—	46	7.6	8.8
Prostate	62	20.9	20.3	—	—	—
Testis	8	.9	.7	—	—	—
Bladder	31	6.8	7.3	8	2.2	2.1
Kidney/Ureter	15	2.7	4.6	4	.6	2.1
Eye	2	.3	.2	2	.2	.1
Brain/Nervous System	45	4.3	4.9	25	4.2	3.3
Thyroid Gland	—	—	.4	1	.2	.5
Thymus/Endocrine	3	.3	.3	—	—	.2
Non-Hodgkins lymphoma	23	3.6	6.0	10	1.8	4.1
Hodgkin's lymphoma	7	.5	1.6	4	.6	1.0
Multiple Meyloma	18	4.9	2.7	9	2.7	1.8
Leukemias	33	4.7	9.1	44	6.9	5.4
Unspecified	71	13.8	12.8	65	12.5	10.1

* indicates cancer death rates in Alaska significantly exceeded those for entire U.S.

Table 2
Cancer Mortality in Alaskan Non-White

Cancer Site	Males Alaska Deaths	Males Alaska Rate	Males U.S. Rate	Females Alaska Deaths	Females Alaska Rate	Females U.S. Rate
All Combined	315	177.4	231.6	248	153.4	133.1
Lip	—	—	.0	—	—	.0
Oral/Tongue	3	1.4	6.5	1	.9	1.7
Salivary Gland	1	.5	.3	3	.8*	.2
Nasopharynx	8	3.5*	.6	5	2.6*	.2
Esophagus	6	3.8	12.3	2	1.2	3.0
Stomach	24	13.9	15.8	9	5.2	6.9
Large Intestine	29	16.9	16.2	34	23.4*	14.9
Rectum	14	7.5	5.0	7	5.3	3.2
Liver/Biliary	22	11.4*	6.9	13	8.5*	3.7
Pancreas	20	12.2	11.8	9	5.7	7.7
Nose/Nasal/Sinus	—	—	.5	—	—	.2
Larynx	4	1.9	3.7	—	—	.5
Trachea/Lung	75	43.4	68.6	16	9.4	13.5
Bone	1	.4	1.0	—	—	.6
Conn/Soft Tissue	1	.6	.9	4	1.7	.9
Melanoma	2	.6	.5	1	.7	.3
Other Skin	2	1.3	.5	—	—	.4
Breast	—	—	.4	27	13.4	22.5
Uterus	—	—	—	3	.8	6.5
Cervix	—	—	—	13	6.9	10.2
Ovary	—	—	—	8	4.8	6.3
Prostate	10	6.5	34.3	—	—	—
Testis	1	.2	.2	—	—	—
Bladder	3	1.5	5.3	2	1.8	2.7
Kidney/Ureter	10	5.7	3.2	8	4.6*	1.5
Eye	—	—	.1	—	—	.1
Brain/Nervous System	4	1.5	2.5	3	.7	1.6
Thyroid Gland	—	—	.3	1	1.0	.5
Thymus/Endocrine	1	.4	.2	—	—	.1
Non-Hodgkins lymphoma	3	1.9	3.8	4	3.2	2.2
Hodgkin's lymphoma	1	.2	1.1	1	1.1	.5
Multiple Meyloma	7	3.3	4.6	4	2.1	3.1
Leukemias	8	3.3	6.4	4	1.9	4.0
Unspecified	55	33.5*	18.1	66	45.7*	13.5

* indicates cancer death rates in Alaska significantly exceeded those for the entire U.S.

Attention Health Care Professionals: Infectious/Contaminated Waste Affects Everyone

The quality of health care in the Anchorage area is a concern to all of us. One of the biggest concerns is the disposal of infectious/contaminated wastes. What do you do with it? Most methods are costly and difficult for small labs and single practitioners. We would like to take this opportunity to introduce our company and the unique service we provide in the Anchorage area to deal with this problem.

New regulations by the Department of Environmental Conservation and the Municipality of Anchorage will soon be in effect regarding the definition and handling procedures for infectious/contaminated wastes. These regulations are necessary to curtail the large influx of potentially infectious material into the landfill. Until recently, most of this material has been going along with your regular trash to the sanitary landfill where it has been exposed to all of the elements — birds, dogs, cats, rodents, insects, etc. This type of material has a potential for carrying pathogenic organisms which ultimately could create a hazardous situation for the general public.

Sure-Way Incineration has designed a program to fulfill the DEC and MOA guidelines. We offer physicians, dentists, hospitals, labs, and other health care professionals a way to properly dispose of infectious/contaminated wastes at a reasonable cost.

By dealing with a firm such as Sure-Way Incineration, the infectious or contaminated wastes are being disposed of properly, thereby relieving you of the responsibility of processing and disposing of the wastes. It now becomes our responsibility.

All infectious or contaminated material is placed in polyethylene containers supplied by Sure-Way. The containers have tight fitting lids to contain any airborne or odor contamination and are labeled "contaminated." The container is replaced each pick-up by a clean, disinfected container on at least a weekly basis.

We feel that this is a very unique service we are offering to the medical profession in the Anchorage area and would appreciate the opportunity to help you meet your needs in this area. If you have any further questions, please feel free to contact us.

**SURE-WAY INCINERATION,
INC.
344-1535**

GEORG STELLER: FIRST PHYSICIAN IN ALASKA

O.W. Frost, Ph.D.[1]

Our first physician in Alaska was a good doctor by almost any standards. In Alaska he faced incredible handicaps, but despite these, he also had some incredible successes. His is a great and inspiring story—and one that should be much better known.

Georg Steller usually isn't thought of as a physician. Some plants and animals, among the hundreds that were first described by him, are today named for him—for example, the Steller jay, the Steller sea lion, the Steller eider, and the Steller sea cow. His first highly competent biographer, Leonhard Stejneger, a noted biologist of the Smithsonian Institution for 60 years, calls Steller "The Pioneer of Alaskan Natural History" in the title of his biography published by Harvard University Press in 1936. In featuring Steller as a naturalist, Stejneger and others tended to overlook Steller's important work as a physician.

How did Steller become a physician, how did he get to Alaska, and what did he accomplish in medicine?

Steller belongs to the first half of the 18th century. His story begins in Germany where he was born in 1709 in a small city named Windsheim in Franconia, not far from Nuremburg. He came from a good, modest, middle-class home, and he was such a good student that citizens of Windsheim awarded him a scholarship to attend the University of Wittenberg. The expectation was that he would study theology and return to his hometown someday to be a Lutheran minister for the rest of his life. After two years of college, a terrible fire intervened. It destroyed much of Windsheim and terminated his scholarship. Stellar was on his own. He shopped around, visiting three universities before he settled on the University of Halle. No doubt his brother, six years older and already a physician from Halle, helped him out.

Halle was a relatively new university and the seedbed of a movement in the Lutheran Church known as Pietism. There were those in the church who felt that too much emphasis was being placed on doctrinal belief and not enough on how one was to practice one's faith. August Hermann Franke, the co-founder of the movement, came to Halle as a professor of theology. He practiced his faith, not only by teaching in the University, but also by founding an orphanage and an orphanage school. He and his colleagues also made Halle a mission center for graduates headed for service in the vast Russian empire under Peter the Great. Graduates were not so much missionaries for the spread of the Lutheran faith, but civilizing agents—counselors, physicians, and professors—in a vast new enterprise to assist Peter the Great in bringing Russia into a modern age of science in navigation, mathematics, cartography, biology, and medicine.

By the time Steller arrived, Halle was already regarded as second only to Berlin as a leading university. It had attracted some great professors of theology, philosophy, and medicine. The orphanage and school had more than two thousand youngsters and was well known throughout Germany. The ties to Russia were well established. Laurence Blumentrost, formerly a Halle medical student, was personal physician to Peter the Great and his chief counselor in founding the first Imperial Academy of Sciences at St. Petersburg. Daniel Messerschmidt, a Halle M.D., became the first trained scientific explorer in Siberia. He was contracted to study the area's geography, natural history, medicinal plants, epidemic diseases, and, if that were not enough, "everything deserving attention"!

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These were no ordinary medical doctors. Blumentrost's father was himself a German physician who had joined the Russian service in 1667. Consequently, the younger Blumentrost, born in Moscow, learned Russian as well as Latin, Greek, and modern European languages before he came to Halle—and later Oxford and Leyden—for medicine. Back in Russia, he became, in 1725, the first president of the Imperial Academy of Sciences.

Messerschmidt was an accomplished cartographer and a speaker of several Oriental languages. After ten years of research in Siberia, 1720-1729, he earned a reputation as the founder of Siberian geography and ethnography. It is to Messerschmidt that Steller has two extraordinary ties: both had the same famous professor of medicine at Halle, Friedrich Hoffmann, who championed them both. The second is even more wonderful; in Russia, Steller married Messerschmidt's young widow!

At Halle, Steller got free board by teaching in the orphanage school and earned a little money by giving a series of private lectures. While continuing his theological studies, he concentrated increasingly on anatomy and botany until finally Hoffmann advised him to go to Berlin to be examined by Professor Michael Ludolf of the medical board. Hoffmann promised to help get Steller a chair in botany at Halle. Steller went to Berlin with the expectation of returning to Halle to be a professor for the rest of his life.

Steller passed the examination, but a professorial appointment became impossible because the King of Prussia, who passed on all such appointments, was at this time deathly ill. Low on funds, Steller could not wait. He put an alternate plan into immediate action. Instead of returning to Halle, he set out for Danzig and St. Petersburg to seek his fame in Russia, and his medical training helped get him there. At Danzig, he came upon a war of Polish succession and was hired on as a surgeon aboard a ship carrying wounded Russian soldiers on the Baltic Sea from Danzig to St. Petersburg. Once in the Russian capital, he was hired by the Archbishop of Novgorod, the head of the Russian Orthodox Church, to be physician to his household staff.

Just how did Steller meet the Archbishop? The usual story—one that Stejneger suggests—is that, just off the boat, Steller headed for the city's botanical garden, the same place where the Archbishop, old and ailing, took his daily constitutional. Supposedly, they bumped into each other. Steller said something cheery like, "Hi, there, old chap!" and the Archbishop, immediately taken with this bright, penniless scholar, invited him to his residence for bed, board, and job.

It is far more likely that Steller went directly to the Archbishop and presented him with a letter of introduction from the son of August Hermann Franke. Once again, Halle was sending out one of its brightest students for service to the Russian Empire—the Archbishop's residence was only a way station toward that end. Certainly, Steller's duties as physician were light enough that he was free to assist Professor Joseph Amman in setting up an herbarium in the new Academy of Sciences and to visit Dr. Messerschmidt, now living in retirement in St. Petersburg.

Steller, at the same time, let it be known that he was interested in joining the scientific contingent of the Great Northern Expedition—that tremendous government-sponsored venture to chart the Arctic coastline of Siberia, to launch voyages of discovery to America and Japan, and to inventory plants, animals, and minerals of Russia's eastern frontier. The Academy had sent out three professors (one French and two German) for scientific exploration as early as 1733, the year before Steller arrived in St. Petersburg. By 1737, the Archbishop and Dr. Messerschmidt had died, and almost simultaneously, the Academy received a plea from Professor Johann Georg Gmelin for assistance in Siberia. Steller was appointed, he married the widow, and off they went in early 1738 from St. Petersburg overland for the Pacific. His new wife did not get very far—only to Moscow—when she realized that winter travel on sledges and the prospect of years of wilderness living in desolate Siberia were too horrible to contemplate. So here they parted, never to see each other again. Having lost a new wife, Steller was later to write that instead of loving a woman, he "fell in love with nature."

And that he did, with a zest and commitment Gmelin found amazing. Gmelin traveled about in style with a large retinue of assistants, servants, and cooks, a small library, sizable wardrobes, and fine wines. He was dumbfounded that, for Steller, any boot or coat would do, and that Steller prepared his own simple meals, using the same spoon and cup for everything. Gmelin saw in the summer of 1739 that nothing could discourage Steller's botanizing around Lake Baikal; he collected and described a thousand and more plants. Gmelin realized, with great relief, that Steller was clearly the one to go on to Kamchatka on the Pacific seaboard, a destination for the scientists as well as for Vitus Bering and his naval officers. In ten years of lumbering around Siberia, Gmelin never got to Kamchatka.

Steller had what he called "an insatiable desire to visit foreign lands"—and he also had his own private agenda. Even before he arrived on Kamchatka, he told Captain Spangberg, whom he met on the way, that he could be his physician on the next voyage to Japan. When he later met Bering, Steller volunteered his services for a first voyage to America. Even before Bering invited him to go, Steller made certain preparations. He asked the aborigines and Cossacks who had traveled throughout Northeast Asia, "In what direction and how far away is America?" And as Bering's future physician, he asked himself an equally important question, "Why do Russians get scurvy and native inhabitants do not?" A simple question. And before he got on Bering's ship, the St. Peter, a year later, he thought he had the answer. It was question of diet. The Kamchadals, he found, had remedies for practically everything. They had what Steller called "scurvy berries"—the yellow cloudberry, the red lingonberry, and the black crowberry. They also consumed, with dried fish as the staple of their diet, many plants including the saranna lily, sweet grass, and what today is called scurvy grass.

On the eve of departure, Bering's physician, pleading illness and exhaustion, begged off making the voyage. Bering also lacked a mineralogist, called for in his instructions. Steller could meet both requirements. Actually, in Steller, Bering got far more than he bargained for. Steller had both the education and experience to be his physician. As a trained natural scientist, Steller was familiar with minerals as well as plants and animals. And as a young man who grew up to prepare himself for the ministry and ended up at the University of Halle, Steller was now—unofficially—a pastor for any good cause. As physician and naturalist, he was constantly on the alert for new theories and specimens. And as Halle had later inspired both Moravians and Methodists (who also ended up in Alaska to build orphanages, schools, and hospitals), so Steller, as a man of Halle, was very much a modern pilgrim in the North Pacific who sought, above all, to work for the common good of what he perceived to be Bering's worthy enterprise. Yet he found himself repeatedly rejected and even ridiculed by those in authority around him.

But Steller's is not a sad story. Only as long as seamen were able-bodied and the ship seaworthy was Steller largely ignored. He was constantly reminded that his opinions had no validity because he was "no seaman." But once the triple disasters of scurvy, storms, and shipwreck overwhelmed the St. Peter and spilled its survivors upon the shores of an unknown and uninhabited island, Steller was quickly recognized for what he had to offer all along—quiet but decisive leadership, healing art, and humility, courage, and wisdom. In the end, then, the journal he kept during the voyage is a record of a personal triumph and fulfillment. But because of his modesty, even in his journal, his role in the deliverance of 45 survivors of an original crew of 78 can be easily overlooked.

While on the St. Peter, Steller's problem, from a critic's point of view, was basically one of his own making: he would not mind his own business. For example, at 52 degrees latitude (under today's Aleutian Islands) he recommended to Bering and other naval officers that the ship be turned northward where all signs of land were pointing. The fact that his advice was usually sound did not matter. He was bucking the system, ignoring the naval chain of command, and as a physician and scientist, he was an outsider not to be included in sea councils where important decisions were made. He was certainly not to be consulted on nautical matters.

As I read him—after having translated his manuscript journal with the invaluable aid of my colleague, Dr. Margritt Engel—Steller was a universal man for whom everything was his business. But he was no elitist. Very much a democrat, his ideal was an enterprise in which everyone worked with a single will under firm leadership toward a common goal. For Steller, Bering did not usually provide that leadership. In sea councils he "needlessly" allowed himself to be outvoted by his own officers.

Once America had been sighted, Bering decided (for once, without a sea council) to look the new country over, to take on fresh water, and to be gone. It is hard to believe that he really intended to deny Steller the opportunity to go ashore to investigate minerals, flora, and fauna. But whatever Bering intended, he did not foresee the intensity of Steller's feelings when, probably for the first time in the presence of his officers, Bering (no doubt teasingly) took Steller on by pretending to "scare" him with "some gruesome murder stories" about what happens to explorers captured by savages. Steller was instantly offended (nothing would keep him from his duty), and two sharp minds and strong wills went head to head until Bering momentarily lost control.

Steller tells the story: "But I answered that I had never acted like a woman, nor did I know of any reason why I should not be permitted to go ashore. To get there was, after all, to follow my chief work, profession, and duty. Up to now I had served Her Majesty faithfully according to my ability and was willing to maintain the honor of my service for a long time yet. And I said that if for reasons contrary to the purpose of the voyage I was not to go, I would report such conduct in terms it deserved.

I was then called a wild man who would not be kept from work even by a treat to chocolate which was just then being prepared.

When I realized that I was to be forced against my will to inexcusable neglect of duty, I put aside all respect and uttered a particular prayer by which the Captain Commander (i.e., Bering) was immediately mollified and let me go with the water-carriers without giving me the least help....

Upon my departure from the ship, the Captain Commander attempted to find out if I could take a joke by having trumpets sounded after me. Without thanking him, I took the matter the way it had been ordered. But I have never been one to blow my own horn and would not have appreciated it if trumpets had been sounded in my honor."

Some readers have interpreted "a particular prayer" to be a euphemism for an oath. But I take Steller literally—for two reasons. Whatever the provocation, Steller was not likely to resort to cursing. He heard plenty of it aboard ship, and he did not approve. Second, Steller was too smart to play into Bering's hands. Were he so gross as to curse his Captain Commander, he could most certainly expect to be put in chains.

Steller, I believe, in his "particular prayer" was making another end-run to higher authority. He had already threatened to report Bering's conduct "in terms it deserved" to the Governing Senate, but Bering did not back down. I suspect he now turned his back on Bering and petitioned Almighty God with a fervor and sincerity no spectator could doubt. Whatever their behavior, Bering and his seamen were devout believers: God did intervene in the world, and God would decide if they, singly or collectively, would live to return home. So not only did Bering lose control of his sideshow with Steller for the entertainment of the crew, but through Steller's prayer he would catch any inference of disobedience concerning a part of his instructions from the monarch who represented God's will in all of Russia.

One can imagine that Bering would break in and say, "Go, go, for the love of God, be off with you!"

To which Steller might have meekly asked for several helpers, only to hear a thunderous "No!"

So Steller got his way to do his duty but at terrible personal cost, for the trumpets marked the end of any favor he had with Bering and signaled to his officers that the Captain Commander had at last turned against the wise guy he kept in his cabin.

They did abuse him just about any way they wished—told him "graciously" later that he'd better get his "butt" off the island and to the ship pronto or they'd leave him stranded, and then refused to let him pack on board from Kayak Island his newly discovered precious salmonberry (another "scurvy berry"). Steller here observes that "as a protestor" he himself now took up too much space. At the only other water stop—in the Shumagin Islands—he was spurned again when, in his proper role as physician, he warned that the water being collected was salty and therefore debilitating and, with a scurvy epidemic breaking out upon them, the crew refused him help to gather plants that he could use for the saving of them all.

Steller, however, proceeded to recruit his best friend aboard ship, German-born Friedrich Plenisner, a surveyor, and his Cossack helper Thoma Lepekhin, probably still a teenager. The three of them spent the day, ranging five miles and more over the south end of Nagai Island, gathering crowberries, lingonberries, dock, and scurvy grass, and no doubt stuffing themsleves with this "medicine" much of the time. It is probably no coincidence that these three men were virtually alone among 75 remaining men in escaping scurvy during the next two months.

Back on ship, Steller, the physician, administered doses of the plants to the sick. He writes in his journal: "Ungrateful and coarse men though they were, my ministrations, under divine grace, very clearly caught their attention when, simply by giving him raw scurvy grass I managed to bring the Captain Commander—so bedridden with scurvy that he had already lost the use of his limbs—so far within eight days that he was able to get out of bed and on deck and to feel as vigorous as he was at the beginning of the voyage. Likewise, the dock I prescribed to be eaten raw for three days firmed up again the teeth of most seamen."

Steller made believers of them all. Five days later, on September 4, 1741, when first contact was made with nine Aleut adults off Bird Island in the Shumagins, Bering and his second-in-command, Lt. Waxell, actually proposed that Steller go with Waxell and a contingent of ten other men to accept an Aleut invitation made by gestures to have food and drink ashore on the island. What a change in the standing of our physician!

The scurvy returned with a vengeance as autumn storms buffeted Bering's ship. By October, says Steller, "people's minds...became as loose and unsteady as their teeth from scurvy." By November men were dying from scurvy daily, and there were only a few men (including Steller) with strength enough to raise and lower sail. On November 5, land was again sighted. "The half-dead," says Steller, "crawled out to see it." Everyone was happy and felt like celebrating. It had to be the Kamchatkan coast, and the decision was made to beach the ship and to send for horses.

Once ashore, parties sent out all came back. There was no escaping a large, uninhabited, treeless island! By January, 32 out of 78 men had died of scurvy, including Bering, but the fresh meat from sea mammals, chiefly sea otters, held further deaths in check. The following spring when snow had melted, Steller again gathered scurvy berries, scurvy grass, and other plants. Then everyone was wonderfully fit again.

Specifically, Steller fed the survivors:

Kamchatkan sweet grass (Heracleum lanatum)
sarana lily bulbs (Fritillaria camschatcensis)
hemlock parsley (Conioselium chinense)
wild celery (Angelica lucida)
oysterleaf (Mertensia maritima)
fireweed (Epilobium angustifolium)
bistort (Polygonum viviparum)
lingonberry (Vaccinium vitis idaea)
scurvy grass (Cochlearia officinali artica)
brooklime (Veronica americana)
bitter cress (Cardamine beringensis)

These plants are mostly old friends Steller learned to use for drink, salads, and vegetables by imitating the example of the Kamchadals.

Not everyone would take their medicine. Bering, for example, would eat only ptarmigan. One day Steller offered to cook a sea otter pup for his very sick Captain Commander. Bering would have none of it, saying to Steller, "I wonder at your taste." Steller replied gently, "I adapt as changing circumstances require." That simple statement is surely a recipe for survival anywhere anytime.

Certainly the medicine chest on the St. Peter was totally useless. Steller tells us it had almost nothing but "plasters, ointments, oils, and other surgical supplies needed for four to five hundred men with wounds from great battles but nothing whatever needed on a sea voyage where scurvy and asthma are the chief complaints."

A genuinely modest man, Steller does not tell us in his journal that, once some 900 sea otter pelts were divided, he wound up with nearly a third of them, many as gifts of gratitude from his companions. The source of that information is Gerhard F. Mueller, who interviewed survivors before writing a history of the Bering voyages.

It is significant that when the first exaggerated newspaper reports about Bering's death reached Steller's family in Germany in 1743, Steller was extolled, not for having overcome scurvy, but for having assumed Bering's role as leader and for having, with the aid of others, rebuilt the small ship. Steller wrote his older doctor brother, "I should like very much to know who has been making me out a seaman and a windbag. My desire is to fill gaps in the realm of science, not vacant space in newspapers." Because of his pledge of secrecy to the Russian state, Steller was not at liberty to tell his brother what he had done or even to correct newspaper reports. Nowhere does he write, "In 1741-1742, I made an important medical discovery."

But thanks to native peoples living on Russia's Pacific seaboard, Steller did make a medical break-through twelve years before Dr. James Lind, "the father of nautical medicine," published his landmark **Treatise of the Scurvy** in 1753. Lind's research, largely ignored when published, was occasioned by the disastrous round-the-world voyage of Lord Walter Anson during 1740-42, when 1,051 out of 1,955 men on the three ships of Anson's fleet died, practically all from scurvy. It is no wonder that Bering and his officers ignored Steller's plea for help in collecting antiscorbutic plants in the Shumagins. They had no idea that scurvy could be treated. It was simply a horror to be expected on long voyages. The best one could do was to keep dry, keep moving, and to let the ship's physician or surgeon lance some blood from time to time.

Lind conducted clinical tests based on anecdotal information about the juice of lemons and oranges as remedies for scurvy. His tests demonstrated the efficacy of these remedies. Captain James Cook had apparently not read Lind's research when he set sail on his first voyage in 1768, but he did have his own lemon juice and was prepared to test a variety of supposed remedies. Unfortunately, the physician who was to conduct the tests died of dysentery enroute, and he left no written results of tests. Still, Cook on all his voyages had a remarkable record in combatting scurvy.

As Wayne W. Myers has written, in **Exploration in Alaska,** published by the Cook Inlet Historical Society in 1980, "(Cook) seemed to feel that if (a food) were green, it would probably be good for you." Cook was the first sea captain to forage for vegetables whenever landings were made. However, it was not before 1795 that regulated spoonfuls of lime juice became standard practice in the British Navy, giving us the word "limey," and scurvy on long sea voyages henceforth came to an end. It was not, of course, until 1932 that the cause of scurvy, as a vitamin C deficiency, became fully understood.

Steller's contribution, though well documented in his manuscript journal of 1743, was certainly an isolated case. His journal was not first published in any form until 1793. Even Lind's treatise of 1753 was not acted upon until 1795. If Cook conquered scurvy, so did Steller—and a generation earlier. If Lind and Cook learned from hearsay dating back to the 16th century, Steller learned from people who really knew what to eat for their good health. Certainly, to this very day, no one has given Steller credit for deserving even a footnote in the history of medical discoveries.

But Steller on Bering Island needed more than a cure for scurvy. These were the circumstances under which Steller worked. First, it was scurvy—abetted by bad water, by diminished stores of biscuit, flour, groats, salted beef, and brandy, by the terror of storms of hurricane force, by exposure to a cold, wet winter coming on, and by the accumulating filth and lice on a ship no longer (80 feet) than many an Alaskan coastal fishing boat today. Second, it was the slow realization that they had happened upon an undiscovered island and not upon Kamchatka. How could anyone survive?

It is possible to read all Steller's journal and still not realize that Steller was effectively in charge during the first two months on the island. He cared for the sick and depressed and also instituted the democratic regime for which he had vainly yearned aboard ship. Consider: Bering and his top officers remained on board the St. Peter in the shallows of the bay. From the first day, November 6, Steller, with two assistants, remained on shore as the sick were carried to open prepared sandpits which Steller cheerily called "graves." On November 9, Bering himself was carried out of his cabin on a litter and deposited in one of these holes. His scurvy was so far advanced that he could scarcely eat or talk. Finally, on November 21, the lieutenant and fleet master, both gravely ill, were among the last to be removed from the ship. Who was in charge, organizing sandpit households, hunts for fresh meat, foraging for driftwood, cooking, fending off Arctic foxes, and nursing the sick?

Nearly everyone was sick, feeble, cold, wet, and deeply depressed. Steller very deliberately took up what might be called pastoral or psychological counseling. He started with the young man he had brought along to be his helper and who now understandably blamed him for his sorry lot.

Steller quotes the conversation he had with him: "Cheer up! God will help. Even if this is not our country, we still have hope of getting there. You will not die of hunger. If you cannot work and wait upon me, I will wait upon you. I know your honest heart and your merit on my behalf. Everything I have is yours also. I will share half with you until God helps."

"Good enough," is the reply. "I will serve (you) with pleasure. But you have brought me into this misery. Who forced you to go with these people? Could you not have had your share of the good things of life on (Kamchatka)?"

Steller must have laughed goodnaturedly at such candor, for he said: "Thank God, we are both alive! If I have dragged you into this misery, then you have also, with God's help, a faithful friend and benefactor in me. My intentions were good, Thoma. So let yours be good also."

The historian Mueller wrote that, during the ten-month period of being stranded of Bering Island, the men "could not lose heart, because they had Steller with them. Steller was a doctor who at the same time ministered to the spirit; he cheered everyone with his lively and agreeable company." So the good doctor who for three months at sea was truly a suffering servant had become, by the end of the voyage, someone resembling a recognized saint. The man of conscience who would not keep in his place proved himself to be, in the fearful, unknown world of the North Pacific, a good example of Emerson's "Man Thinking," alert to human need as well as to signs in Nature and ever ready to reason, comment, and take action.

Steller's knowledge of science was broad and practical. He saved many lives. He was a good man. He is a physician to honor. That he died in 1746 before he had published any of his manuscripts is sad indeed. That his achievements, other than in biology, have been so insufficiently recognized is, I believe, and injustice still begging for correction.

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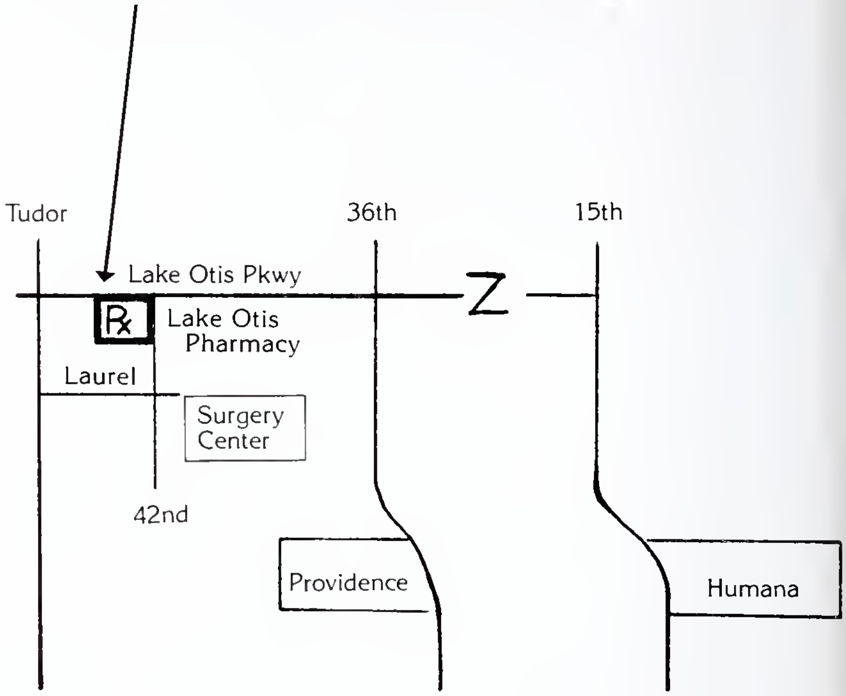

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ESTIMATES OF CANCER INCIDENCE IN ALASKAN NATIVES DUE TO EXPOSURE TO GLOBAL RADIOACTIVE FALLOUT FROM ATMOSPHERIC NUCLEAR WEAPONS TESTING

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Abstract

During the peak period of atmospheric nuclear weapons testing in the northern hemisphere in the early 1960's, measurable concentrations of cesium-137 and strontium-90 accumulated in native residents of certain northern Alaskan villages through the lichen-caribou food pathway. Now that a significant latent period for cancer induction has elapsed, the question of possible cancer increases from this radiation exposure has been raised. To address this question, radionuclide measurement data and dose estimates made during this period were reviewed. Leukemia, breast cancer, and bone sarcoma were identified as the malignancies most likely to be induced from internally deposited cesium-137 and strontium-90, and risk estimates were developed for these cancers. Maximum annual dose rates due to these radionuclides were found to be low and comparable to the natural background radiation levels that exist in certain parts of the United States. In addition, the number of Alaskan natives likely to have received these maximum doses was found to be very small. As a result, the number of cancer cases expected from this exposure is too low to be detected by epidemiologic study of the populations actually exposed. Fallout radionuclides other than cesium and strontium have also been detected in Alaskan ecosystems, but at levels resulting in doses considerably lower than those referred to above.

Abbreviations

Ci — Curie (a measure of the quantity of radioactive material)
mCi — millicurie (10^{-3} Ci)
uCi — Microcurie (10^{-6} Ci)
nCi — nanocurie (10^{-9} Ci)
pCi — picocurie (10^{-12} Ci)
rad — A measure of radiation dose in tissue
mrad — millirad (10^{-3}rad)
rem — a measure of radiation dose equivalent, an entity which takes into account the quality of the radiation as well as the absorbed dose in rads. The rem is equivalent to the rad for gam-

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ma and medium to high energy beta radiation.

mrem — millirem

Introduction

During the early and middle 1960's, radionuclide body burdens were measured in a considerable number of northern Alaskan Natives because of concern about concentration of radioactive fallout through the lichen-caribou-human food pathway. Now that a significant latent period for cancer induction has passed, recent public concern has been raised by Natives about possible increases in cancer incidence among Alaskan Natives as a result of this exposure. To address this concern, personnel from the Cancer Branch, Center for Environmental Health, Centers for Disease Control (CDC) met with personnel from the Arctic Investigations Laboratory, Center for Infectious Diseases, CDC, Anchorage in August, 1984, to review the problem. Several approaches to the problem were planned: 1) to assess the original methods of measuring radiation exposure and the estimates for the groups exposed; 2) to calculate the cancer incidence which could result utilizing estimates of dose and population exposed in Alaska; 3) to review Alaska Native Tumor Registry data pertinent to the radiation issue; and 4) to obtain original measurement data on individuals and compare this with their subsequent cancer experience. This report includes items 1 and 2 above; items 3 and 4 will be reported subsequently.

Background

The bulk of atmospheric nuclear weapons testing was carried out from 1945 to 1963 in the northern hemisphere by the U.S.A. and the U.S.S.R. During this period, 379 atmospheric tests were conducted. In the ten years following the Limited Test Ban Treaty in 1963, only 43 atmospheric tests were conducted, 29 by France and 14 by the People's Republic of China, neither of whom were party to the treaty (1). Although subsurface nuclear weapons testing by the major powers has continued since 1963, the peak periods of contamination of the biosphere, documented by extensive radiation monitoring throughout the world, occurred during the 1950's and the early 1960's.

Radioactive fallout consists of radioactive particles that have entered the atmosphere as a result of nuclear detonations. Explosions at, or slightly above, the earth's surface (i.e. atmospheric nuclear tests) result in the greatest release of radionuclides into the atmosphere and, therefore, the largest amount of fallout. Subsurface nuclear detonations (the predominant type after the test ban treaty) release only a fraction of the total resultant radioactivity, that fraction being inversely related to the depth of the detonation.

Radioactive fallout is classified as local or global depending on its spatial and temporal distribution. Local fallout consists of larger particles (generally over 35 microns in diameter) and falls back to the earth's surface within about a day and within several hundred miles or less from the detonation site. Global fallout is composed of smaller particles, rises higher into the atmosphere, and becomes widely dispersed over the earth's surface. The higher the yield of the nuclear device (i.e. the megatonnage), the higher the fallout particles are blown up into the atmosphere, and the longer they take to return to the earth's surface.

The atmosphere can be divided into the troposphere (from sea level to about 45,000 feet) and the stratosphere (from over 45,000 feet to about 160,000 feet). Low yield detonations yield mainly tropospheric fallout which returns to the surface within a month or so from the time of detonation. Fallout deposition from the troposphere is dependent on weather conditions and usually occurs along a band in the same latitude as the detonation site. High yield detonations are powerful enough to push radioactive material up into the stratosphere. This material returns to the surface over a period of months to several years. Deposition is a function of latitude and deposits occur in higher concentrations in the temperate zones. Since the majority of the weapons testing has taken place in the northern hemisphere, more fallout occurred in the northern than southern temperate zone. The major portion of global fallout from nuclear weapons testing has been from the stratosphere.

Fallout is composed of a variety of radionuclides whose biological importance depends on factors such as the type of radiation emitted, particle size, solubility, physical and biologic half-life, etc. Only a few of these radionuclides pose a potentially significant long-term health hazard since only some occur in abundance, have relatively long half-lives, and have chemical characteristics that facilitate transport and concentration through food chains and result in accumulation of significant amounts of radioactivity in sensitive body tissues. Of the 200 or more radionuclides possibly present right after a nuclear explosion, only a small percentage have half-lives long enough that they exist more than a few hours. Since most global fallout has been stratospheric, which takes months to years to return to the earth's surface, very few of these nuclides are deposited as radioactive fallout. Of those that are, even fewer are found in concentrations sufficient to pose a potential hazard to human health. For this to occur, some type of mechanism of concentration through the food chain is required. The major factor involved in the concentration of fallout radionuclides in arctic and subarctic food chains is the ability of the lichen to absorb and retain particles from the atmosphere and from precipitation. A large fraction of fallout material can be directly absorbed by the lichen and, because lichens have evolved very efficient mechanisms for conserving nutrients in their barren environments, much of this is retained in the plant for many years. Northern Alaskan caribou migrate southward in the fall into the Kobuk and Koyukuk River drainage areas, where they spend the winter and feed predominately on lichens. In the spring the caribou migrate northward back through the passes of the Brooks Range, and during the summer wander throughout the north western Arctic feeding predominately on seed plants, which have fallout radionuclide concentrations two to ten times lower than those found in lichens.

During the 1950's and early 1960's, subsistence hunting and fishing provided the economic base for the northern Alaskan native villages. These subsistence economies have been gradually shifting toward capital-based economies because of the increasing immigration and influence of white people in northern Alaska, particularly in the larger coastal villages. This shift was accelerated by the oil industry development that occurred in the North Slope region during the 1970's. One result of this shift has been a general decrease in dependence on caribou as a food source. However, during the period of maxium fallout and continuing through the 1970's, caribou was a major food source for many of the northern villages. This was particularly true for the Eskimos of Anaktuvuk Pass in the Brooks Range, where the highest body burdens of cesium-137 occurred in 1964 (2).

Cesium-137 has a radioactive ("physical") half-life of 30 years (see Table I) and a biochemical behavior similar to potassium so that it accumulates and concentrates in many different body tissues, including caribou muscle which may be eaten in large quantities by certain Native groups. After ingestion, cesium is rapidly absorbed and, since it is soluble in body fluids, it is distributed fairly uniformly throughout the body, although concentrations are generally higher in muscle than in bone and fat. The biological elimination half-time is about 135 days in adult males, 85 days in adult females, and ranges from about 60 days in older children down to 12 days in infants (3). Thus, for a given initial body burden, dose would be roughly proportional to body mass (i.e., younger, smaller persons would receive a lower dose per unit body burden). Pregnant women also have a shorter biological elimination half-time, resulting in a lower dose per unit body burden to the fetus (4). From the standpoint of carcinogenesis, since cesium distributes fairly evenly throughout the body and emits penetrating higher energy gamma radiation, the critical organs are those that are most sensitive to induction of cancer by radiation, namely bone marrow and breast (5).

Strontium-90 is another fallout radionuclide of potential concern because of its long physical half-life (28 years), its absorption and concentration by lichens, its concentration in caribou bone after ingestion, and its long effective half-life in bone (18 years—see Table 1). Strontium has a biochemical similarity to calcium, which explains its affinity for bone. Because it emits high energy beta radiation, strontium-90 deposited in bone irradiates both the calcified bone and the adjacent bone marrow (3). The tumor types of prime concern with strontium exposure are therefore bone sarcoma and leukemia.

Iodine-131 is also of some possible concern because, although it has a much shorter physical half-life (8 days—see Table I), it emits beta particles and concentrates like ordinary iodine in the thyroid gland, one of the organs most sensitive to cancer induction by radiation (5).

Table I

Metabolism of Selected Radionuclides

Radionuclide	Half-life Physical	Half-life Effective*	Target Organ
Cesium-137	30 yrs	135 days	Whole body (similar to potassium)
Strontium-90	28 yrs	18 yrs	Bone (similar to calcium)
Iodine-131	8 days	7.6 days	Thyroid

* Effective half-life takes into account both the decrease in radionuclide activity due to radioactive decay (physical half-life) and the decrease due to biological elimination of the nuclide from the body (Biological elimination half-time).

Although the three radionuclides just discussed are the primary ones of concern because they may cause long-term health effects, several other fallout nuclides also merit some mention. However, these other nuclides do not appear to be significant long term health hazards for Alaskan Natives because: 1) no effective concentration process occurs in the arctic and subarctic ecosystems that would result in a significant accumulation of, and dose to, humans; 2) their physical half-life is too short to present a long-term hazard, given the time frame over which the accumulation processes occur; or 3) they simply do not exist in sufficient quantities to present a significant health hazard.

The period of greatest global fallout occurred in the early 1960's. In 1965 measurements were made of a number of different radionuclides in lichens, in tissues of caribou and wolves (which feed on Caribou), and in urine samples of Eskimos in the Anaktuvuk Pass region. Results of these measurements are the basis of many of the comments in Table II.

Table II

Selected properties of fallout radionuclides of potential public health importance for Alaskan Natives

Nuclide	Physical half-life	Comments
Tritium (a)	12 years	no concentration process; mean urine concentration in Noatak in 1972 was 1500 pCi/1, corresponding to an annual dose of 0.15 mrem (6); present in form of tritiated water.
Carbon-14 (a)	5,600 years	no concentration process; present in form of carbon dioxide; eventually transferred to deep oceans.
Argon-39 (a)	260 years	inert gas; no concentration process.
Iron-55 (b)	2.9 years	concentrates most in ocean fish (salmon, tuna); Alaskan natives had maximum body burdens of 2.3uCi(7) (maximum permissible body burden for general population is 100 uCi for 55-Fe).
Ruthenium-106 (c)	1 year	no concentration beyond lichen stage of food chain; not detected in caribou flesh, wolf flesh, or Eskimo urine (8).
Cerium-144 (c)	290 days	no concentration beyond lichen stage in food chain (very poorly absorbed from gastrointestinal tract in man); not detected in caribou flesh, wolf flesh, or Eskimo urine (8).
Plutonium-239 (d) Plutonium-240 Plutonium-241	24,400 years 6,580 years 13.2 years	unlike Cs and Sr, Pu is not related to any nutrient element; degree of transport in food chains is very low (successive trophic level concentration factor is less than 0.01) (1); inhalation would be the most important exposure pathway; mean dose commitment in U.S. from nuclear testing through 1970 is 2 mrad to the lung and 0.2 to the bone; doses estimated in Lapps through lichen-reindeer food chain are negligible (9).
Americium-241 (e)	458 Years	primarily created in situ from ^{241}Pu; estimated doses in Lapps negligible (9).

(a) Neutron activation product (air)
(b) neutron activation product (soil)
(c) fission product
(d) may exist as unreacted weapon component
(e) decay product of ^{241} Pu

Dose Assessment

During the 1960's a considerable amount of measurement of radiation was done in the arctic and subarctic ecosystems, including assessments of radionuclide levels in lichens, caribou and Eskimos. Of prime importance are the cesium-137 body burden measurements done during various years on Eskimos in many of the northern Alaskan villages, particularly Anaktuvuk Pass where caribou was the primary component of their diet at that time.

Body burdens of the higher energy gamma-emitting radionuclides such as cesium-137 can be directly measured by whole-body counting. The classical whole-body counter consists of one or more sensitive gamma detectors in a heavily shielded iron room. This arrangement permits quantification and identification (by its specific gamma energy spectrum) of small amounts of a radionuclide in the body, with minimal interference from natural background radiation. Comparison of the amount of activity detected with a known quantity of the same radionuclide measured in a phantom allows an accurate determination of the body burden.

Portable whole-body counters, which require about 5 tons of lead brick shielding, have been used for some cesium-137 body burden measurements in Alaskan Eskimos (10). These counters reduce background about as well as the iron-room counter, show little variation in sensitivity with body size, and are only slightly less sensitive than the iron-room counter. Average body burden measurements are generally within a few percent of the expected values based on counting in iron-room whole body counters. A less cumbersome whole-body counting method, which does not use any lead shielding, has been developed for field measurements (11). A gamma detector is placed in the sitting subject's lap, and the subject is counted while bending over the detector. This configuration gets as much of the subject's body as close to the detector as possible, while the body provides some shielding from background radiation. The counting efficiency decreases with increasing body size and measurements are corrected for this factor. This method is best suited for radionuclides that are distributed uniformly throughout the body, such as cesium, and for subjects with higher body burdens. The accuracy of this technique has been estimated to be ±20% for body burdens around 200 nCi cesium-137 and decreases to ±100% at body burdens of 40 nCi (based on comparison of subjects counted by both this technique and the iron-room whole body counter). Many of the cesium-137 body burden measurements in Alaskan Eskimos were done using this simplified technique, which was appropriate since body burdens of possible health concern are well above 200 nCi.

The highest average cesium-137 body burdens measured (over 1000 nCi) occurred in Anaktuvuk Pass residents in the summer of 1964. From 1962 through 1967, residents of Kotzebue and regional river villages of Noatak, Selawik, Noorvik, Kiana, Shungnak, Kobuk, and Ambler had intermediate levels (150-650 nCi), and the northern coastal villages of Barrow and Point Hope had the lowest levels (3-150 nCi) (12-18). The maximum levels in Anaktuvuk Pass occurred about 2 years after the peak period of atmospheric testing in 1962. This time lag is consistent with stratospheric fallout deposition and subsequent concentration of cesium through the lichen-caribou-human food pathway. Since 1964, cesium levels in Anaktuvuk Pass residents have slowly decreased, as shown by body burden measurements done in the early 1970's (19). The slowness of this decrease is due to the 30-year half life of cesium-137 and the 10-year or longer retention half-time for cesium in lichens. The seasonal fluctuation occurs because the caribou feed mainly on lichens only during the winter. Caribou killed in the spring (and consumed over the summer) had higher levels than those killed in the fall. This resulted in higher summer cesium levels in the Eskimos.

Because strontium-90 is a pure beta emitter, body burdens cannot be measured by whole-body counting techniques. However, strontium-90 body burdens in Anaktuvuk Pass residents have been estimated on the basis of annual strontium ingestion rates and the metabolism of strontium in humans. Strontium-90 concentrations were measured in caribou meat samples obtained from Anaktuvuk Pass Eskimo hunters from 1964 to 1966 and adult male Eskimo body burdens were estimated to be about 900 pCi, which was very similar to body burdens of residents of New York and San Francisco during the same period (20). (Residents of the contiguous 48 states had elevated strontium levels from consumption of dairy products and vegetables, which are not important components of the traditional Eskimo diet). A higher average estimate (7400 pCi) was made from strontium-90 concentrations measured in 9 individual rib samples from Alaskan subjects obtained during 1963 and 1964, although these concentrations were also similar to those in bone specimens from residents of other states (21,22). Strontium-90 ingestion rates, and estimated body burdens, for adult females and for children were, respectively, 50% and 20% of those for adult males. Caribou meat provided 80 to 95% of the strontium-90 body burdens of northern Alaskan Eskimos during the 1960's. The steadily decreasing body burdens since 1966 resulted more from a decreased dependence on caribou as a food source, rather than from decreasing levels of strontium in caribou meat (20).

Although direct measurements of iodine-131 in thyroid glands of Alaskan Eskimos were not done, measurements and dose calculations were done on thyroids from deer, elk, caribou, and reindeer from Alaska and several other states during and after the peak 1962-63 nuclear testing period. Doses received by these herbivores during this period are shown in Table III (23).

Table III

Thyroid doses received by selected herbivores from 131-I fallout during the 1962-63 nuclear testing period.

State	Animal	Thyroid Dose (rem)
Colorado	Deer	20
Wyoming Washington	Elk	7.6
California Maryland New York	Deer	2.5
Alaska	Caribou Reindeer	0.8

The herbivore thyroid dose appears to be related to distance and direction from the sites of testing. This pattern is consistent with the relatively short half-life of the iodine-131 (8 days) and suggests that tropospheric fallout deposition is the predominant process involved. Alaskan herbivores received the lowest thyroid dose of all the animal locations sampled. This suggests that either arctic deposition of iodine was lower or that iodine did not enter the arctic food chain as readily as in other areas of the country. No specific thyroid dose estimates are available for Alaskan Natives. However, dairy products were not an important part of the traditional Eskimo diet, and no other significant human exposure pathway is evident.

Elevated levels of certain naturally occurring radionuclides have also been measured in Anaktuvuk Pass residents. Lead-210 and polonium-210, the solid decay products of radon-222 which occurs naturally in the atmosphere, have been found in relatively high concentrations in arctic lichens and caribou. The concentration process for this "natural fallout" is similar to the one observed with cesium-137 and strontium-90. Concentrations of polonum-210 in caribou flesh were about 10 times greater than lead-210 concentrations. Measurements of polonium-210 in urine samples from Anaktuvuk Pass residents in the early 1960's showed levels 200 times higher than those measured in other states. These levels corresponded to about ten percent of the maximum permissible body burden for polonium-210 (24).

Cancer Risk Estimates

Cancer risk estimates were based on information from the National Research Council Committee on the Biological Effects of Ionizing Radiations, 1980 (BEIR III Report) (5). They are expressed as a range of numbers rather than as a precise value because of the uncertainty associated with carcinogenic risk from radiation. Several important points must be emphasized regarding these risk estimates and resultant expected cancer incidence rates that were derived from them:

1. The highest average dose measurements were used in calculating expected cancer incidence. These occurred in Anaktuvuk Pass residents, who were still largely dependent on caribou for their food source in the early and middle 1960's. The actual number of persons who received these maximum doses was probably less than 100 (the total Alaskan Native population of Anaktuvuk Pass in 1970 was 97) (25). The population in 1980 of the northern Alaskan villages where measurements were taken was 5,715 (26). (The total Alaskan Native population was 64,047 in 1980 and 50,819 in 1970). Thus the percentage of Alaskan Natives with additional radiation exposure from fallout via the lichen-caribou food chain is small.

2. Expected cancer incidence rates were calculated with the assumption that the peak exposure levels of the middle 1960's remained at the same level over the next 20 years. Body burdens have actually been steadily decreasing during that period. Average cesium-137 body burdens in Anaktuvuk Pass residents measured in 1979 result in a dose of 8 mrem per year (2), which is more than 20 times lower than the peak dose levels in 1964. Strontium-90 body burdens have been decreasing by about 9 percent per year since 1970 (20).

3. When risk estimates for a particular cancer site were based on more than one risk model in the BEIR III Report, the highest risk estimates were used to calculate the upper limit of the expected cancer incidence rate.

These three factors result in a "worst case" estimate of expected excess cancer incidence rates, and the "most likely case" estimates may be 10 to 1000 times lower. Expected excess cancer rates

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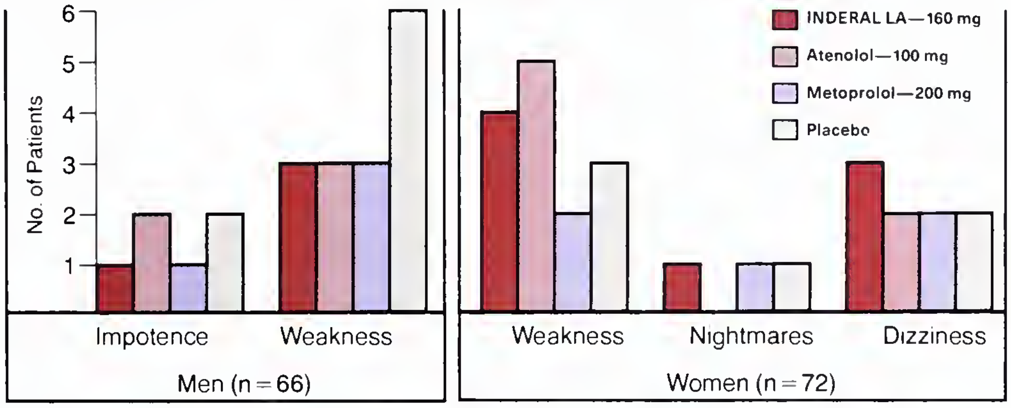

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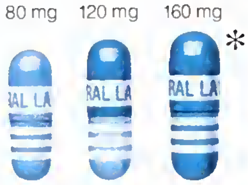

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CONTRAINDICATIONS

Propranolol hydrochloride (INDERAL® LA): Propranolol is contraindicated in: 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with propranolol.

Hydrochlorothiazide: Hydrochlorothiazide is contraindicated in patients with anuria or hypersensitivity to this or other sulfonamide-derived drugs.

WARNINGS

Propranolol hydrochloride (INDERAL® LA): CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated, and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or propranolol should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction following *abrupt* discontinuance of propranolol therapy. Therefore, when discontinuance of propranolol is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when propranolol is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If propranolol therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute propranolol therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD, IN GENERAL, NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution, since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin. Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure.

Hydrochlorothiazide: Thiazides should be used with caution in severe renal disease. In patients with renal disease, thiazides may precipitate azotemia. In patients with impaired renal function, cumulative effects of the drug may develop.

Thiazides should also be used with caution in patients with impaired hepatic function or progressive liver disease, since minor alterations of fluid and electrolyte balance may precipitate hepatic coma.

Thiazides may add to or potentiate the action of other antihypertensive drugs. Potentiation occurs with ganglionic or peripheral adrenergic-blocking drugs.

Sensitivity reactions may occur in patients with a history of allergy or bronchial asthma.

The possibility of exacerbation or activation of systemic lupus erythematosus has been reported.

PRECAUTIONS

Propranolol hydrochloride (INDERAL® LA): GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. Propranolol is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that propranolol may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs, such as reserpine, should be closely observed if propranolol is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity, which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies, in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. Propranolol has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximal recommended human dose. There are no adequate and well-controlled studies in pregnant women. Propranolol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: Propranolol is excreted in human milk. Caution should be exercised when propranolol is administered to a nursing mother.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

Hydrochlorothiazide: GENERAL: Periodic determination of serum electrolytes to detect possible electrolyte imbalance should be performed at appropriate intervals.

All patients receiving thiazide therapy should be observed for clinical signs of fluid or electrolyte imbalance, namely: Hyponatremia, hypochloremic alkalosis, and hypokalemia. Serum and urine electrolyte determinations are particularly important when the patient is vomiting excessively or receiving parenteral fluids. Medication such as digitalis may also influence serum electrolytes. Warning signs irrespective of cause are: Dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, and gastrointestinal disturbances such as nausea and vomiting.

Hypokalemia may develop, especially with brisk diuresis, when severe cirrhosis is present, or during concomitant use of corticosteroids or ACTH.

Interference with adequate oral electrolyte intake will also contribute to hypokalemia. Hypokalemia can sensitize or exaggerate the response of the heart to the toxic effect of digitalis (eg, increased ventricular irritability). Hypokalemia may be avoided or treated by use of potassium supplements, such as foods with a high potassium content.

Any chloride deficit is generally mild and usually does not require specific treatment, except under extraordinary circumstances (as in liver or renal disease). Dilutional hyponatremia may occur in edematous patients in hot weather; appropriate therapy is water restriction, rather than administration of salt, except in rare instances when the hyponatremia is life-threatening. In actual salt depletion, appropriate replacement is the therapy of choice.

Hyperuricemia may occur or frank gout may be precipitated in certain patients receiving thiazide therapy.

Insulin requirements in diabetic patients may be increased, decreased, or unchanged. Diabetes mellitus which has been latent may become manifest during thiazide administration.

If progressive renal impairment becomes evident, consider withholding or discontinuing diuretic therapy.

Thiazides may decrease serum PBI levels without signs of thyroid disturbance.

Calcium excretion is decreased by thiazides. Pathologic changes in the parathyroid gland with hypercalcemia and hypophosphatemia have been observed in a few patients on prolonged thiazide therapy. The common complications of hyperparathyroidism, such as renal lithiasis, bone resorption, and peptic ulceration, have not been seen. Thiazides should be discontinued before carrying out tests for parathyroid function.

DRUG INTERACTIONS: Thiazide drugs may increase the responsiveness to tubocurarine.

The antihypertensive effects of thiazides may be enhanced in the postsympathectomy patient. Thiazides may decrease arterial responsiveness to norepinephrine. This diminution is not sufficient to preclude effectiveness of the pressor agent for therapeutic use.

PREGNANCY: Pregnancy Category C. Thiazides cross the placental barrier and appear in cord blood. The use of thiazides in pregnancy requires that the anticipated benefit be weighed against possible hazards to the fetus. These hazards include fetal or neonatal jaundice, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult.

NURSING MOTHERS: Thiazides appear in human milk. If use of the drug is deemed essential, the patient should stop nursing.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Propranolol hydrochloride (INDERAL® LA): Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia, congestive heart failure, intensification of AV block, hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type.

Central Nervous System: Lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances, hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability; slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation; mesenteric arterial thrombosis; ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis; nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions; psoriasiform rashes; dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes, and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

Hydrochlorothiazide:

Gastrointestinal: Anorexia, gastric irritation, nausea, vomiting, cramping, diarrhea, constipation; jaundice (intrahepatic cholestatic jaundice); pancreatitis; sialadenitis.

Central Nervous System: Dizziness, vertigo; paresthesias; headache; xanthopsia.

Hematologic: Leukopenia, agranulocytosis, thrombocytopenia, aplastic anemia.

Cardiovascular: Orthostatic hypotension (may be aggravated by alcohol, barbiturates, or narcotics).

Hypersensitivity: Purpura, photosensitivity; rash; urticaria, necrotizing angiitis (vasculitis, cutaneous vasculitis); fever; respiratory distress, including pneumonitis; anaphylactic reactions.

Other: Hyperglycemia, glycosuria, hyperuricemia; muscle spasm; weakness; restlessness; transient blurred vision.

Whenever adverse reactions are moderate or severe, thiazide dosage should be reduced or therapy withdrawn.

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were estimated for four cancer sites that have the greatest potential for induction by radiation in this situation.

A. Leukemia may result from cesium or strontium exposure. Radiation-induced leukemia has a relatively short latent period (median 7 to 8 years in the Japanese A-bomb survivors). Latency appears to be shorter in younger age groups and with higher doses. There is a decreased risk per unit of exposure (risk-per-rem) at lower radiation doses. Estimation of the risk from high dose studies (the only type available) may overestimate the risk from lower doses by a factor of 2 to 10. Evidence also suggests a relative risk 2 to 5 times higher among the very young and the very old.

Acute leukemia and chronic myelogenous leukemia are the major types associated with radiation exposure. Chronic lymphocytic leukemia has not been shown to be related to radiation.(5)

B. Breast cancer may result from cesium exposure. The female breast is very sensitive to induction of cancer by radiation. A conserviatve lower limit for the minimum latent period is 5 to 9 years. The maximum latent period is 30 or more years. Latency appears to be independent of dose but strongly dependent on age at exposure. The lower the age at exposure, the longer the latency period tends to be. The occurrence of radiation-associated breast cancer parallels the age distribution of "spontaneous" breast cancer, after a minimal latent period.

The dose-response for breast cancer appears to be linear down to zero dose, i.e. the risk-per-rem is similar for low and high doses. Risk does not seem to depend on dose rate. However, risk may depend on age at exposure, although precise age pattern is not clear. Risk estimates based on the Japanese A-bomb survivors' experience show a 2 to 3 fold higher risk in the 10 to 19 year exposure age group, compared to the 20 to 39 year age group and the 50+ year age group. There is not substantial evidence yet of increased risk for exposure before age 10 years. The risk in the 40 to 49 year exposure age group is slightly negative and the reason for this is not apparent. These variations in risk of breast cancer with age at exposure may be due to changes in tissue sensitivity to radiation carcinogenesis resulting from variations in ovarian function at different ages.(5)

C. Bone cancer may result from strontium exposure. Risk estimates are based mainly on the knowledge gained from studies of exposure to alpha emitters (eg., radium-226), which have a high relative biological effectiveness compared to beta and gamma emitters. This would tend to overestimate the risk from strontium-90, a beta radiation emitter. Radiation-induced bone cancers have shown a latency period ranging from 4 to 52 years. Generally, latency is directly related to the duration of the exposure. Short exposure periods show a peak latency of 6 to 8 years, while continous long-term exposures (which would result from strontium ingestion) show much longer latent periods. The most common types of radiation-induced bone cancers (in order of decreasing frequency) are osteosarcoma, fibrosarcoma, and chondrosarcoma. No cases have occurred in the radium dial painters at doses much below 900 rads. No increase in bone sarcoma was noted in the Japanese A-bomb survivors.(5)

D. Thyroid cancer may result from iodine exposure. The radiation-induced types of thyroid cancer are papillary carcinoma and follicular carcinoma. Anaplastic carcinoma of the thyroid has not been associated with radiation. The minimum latency period is about 10 years. The peak latency period, if one actually exists, is probably from 15 to 25 years. External gamma radiation has a higher carcinogenic risk than internal beta radiation (such as occurs with iodine-131). The reason may be partly because the iodine resides mainly in the colloid of the thyroid follicle and gives a variable beta dose to the sensitive cellular component of the follicle. The risk from iodine-131 is also lower than that from the shorter-lived radioactive iodine isotopes (which are a local rather than global fallout problem), probably because iodine-131 gives a lower dose rate (since it has a longer half-live) and may allow for some type of cellular recovery or repair.

An entity termed "minimal or occult microscopic thyroid cancer" is found at necropsy in 30% of the Japanese population and 15% of the American population. It is felt to have no malignant potential and is not known to be induced by radiation. Therefore, occult carcinoma should not be included with clinical disease when developing or applying radiation risk estimates.

Radiation-induced benign thyroid adenomas occur 3 times more commonly than malignant carcinomas. Other non-malignant radiation effects on the thyroid gland are associated with higher doses than those which induce cancer: acute thyroiditis — 20,000 rads; and hypothroidism (thyroid ablation) — 2,000 rads external or 5,000 rads internal irradiation.(5)

The range of expected cancer which may result annually from fallout exposure in Alaska is given in Table IV and compared with age-adjusted rates for the United States. It cannot be emphasized enough that these are "worst case" estimates, and also that the actual percentage of Alaskan Natives to which these rates might apply is probably extremely small.

TABLE IV - Cancer risk in Alaskan Natives due to radioactive fallout from atmospheric nuclear weapons tests (based on "worst case" estimate - assuming a 20 year exposure at the maximum dose rate)

Radionuclide	Cancer or tumor type	Highest average body burden or tissue concentration	Corresponding dose rate	Risk coefficient (cases per rem per year per million persons)(5)	Expected annual excess cancer rate (per 100,000) for a 20 year exposure	Annual age-adjusted[3] cancer rate (per 100,000)for United States from SEER program 1973-77(29)
Cesium-137	Leukemia	1330 nCi(27)	190 mrem/yr[1]	0.01 to 2.2	0.004 to 0.8	9.8
	Breast cancer	1330 nCi(27)	190 mrem/yr[1]	0.60 to 6.1	0.23 to 2.3	85.4
Strontium-90	Bone cancer	2.8 pCi/gm Ca(20)	12.5 mrem/yr[2]	0.09 to 0.76	0.002 to 0.019	0.8
	Leukemia	2.8 pCi/gm Ca(20)	12.5 mrem/yr[2]	0.01 to 2.2	0.0002 to 0.055	9.8
Iodine-131	Thyroid cancer	unknown	unknown	4	-	4.0
	Benign thyroid adenomas	unknown	unknown	12	-	

1) 1000 nCi cesium-137 gives 143 mrem/yr whole body and average skeletal dose.(4)
2) 1.0 pCi strontium-90 per gram of calcium in bone (pCi/gm Ca) gives 4.5 mrem/yr skeletal dose.(28)
3) Age - adjusted to the 1970 Census population.

Table V shows some representitive doses from various sources for comparison with the doses due to fallout in Alaska. Natural background radiation includes that from cosmic radiation, external gamma radiation from naturally occurring radioactive material in the earth's curst, and radiation from naturally occurring radionuclides found in the body. Average levels in the United States range from 100 to 250 mrem per year (30). Certain areas of the world have unusually high natural radiation levels, averaging as high as 3000 mrem per year and ranging up to 12,000 mrem per year (31). Epidemiologic studies done in some of these regions have not shown increased cancer incidence attributable to these exposures although the number of people exposed was generally only a few thousand. Average annual medical exposure (32) and the current regulatory guidelines are also given in Table V. Doses received by the Japanese A-bomb survivors ranged from 0 to over 400,000 mrem. There is little evidence of excess cancer at doses below 10,000 mrem and most of the excess cases occured at doses over 50,000 mrem in this group.(33)

Table VI shows the maximum expected numbers of cancer cases per year due to cesium-137 and strontium-90 body burdens for the northern Alaskan villages. With a population totaling 5,715 assuming the worst case estimate, a maximum of 3.6 cases of cancer would have developed in 20 years. These numbers are so small that an increase could not be detected by epidemiologic study.

Table V

Average radiation doses from selected sources.

Source	Dose (mrem)
A. Annual natural background radiation (to whole body).	
1. United States	
Colorado	250
Wyoming	245
New York	135
Alaska	130
Georgia	125
Texas	100
2. High Background areas of the world.	
Kerala, India	1500
Minas Gerias, Brazil	2000
Sri Lanka (Granite areas)	3000
B. Medical exposure	
1. Average annual dose per person in United States	60
2. Dose to exposed organs from specific exams.	
Chest X-ray	4 (bone marrow) 19 (lungs)
Upper GI Series	117 (bone marrow) 532 (lungs)
Barium Enema	298 (bone marrow) 48 (lungs)
C. Regulatory guidelines	
1. Annual occupational limit.	5000
2. Annual general population limit.	500
D. Japanese A-bomb survivors.	
Dose resulting in about 50% mortality in 3 to 5 weeks (received by about 1500 survivors of Hiroshima and Nagasaki)	300,000

Table VI

Maximum expected annual number of cancer cases due to cesium-137 and strontium-90 body burdens for northern Alaskan villages.

Village	Anaktuvuk Pass	Kotzebue	Barrow	Point Hope	River Villages	Total
1980 native population	191	1574	1720	434	1796	5715
Leukemia	0.002	0.01	0.01	0.004	0.02	0.049
Breast Cancer	0.004	0.04	0.04	0.01	0.04	0.13
Bone Sarcoma	0.00004	0.0003	0.0003	0.0001	0.0003	0.001

Conclusions

1. Studies over the past 25 years have adequately identified and measured the fallout radionuclides of potential significant health importance in Alaska.

   A. Cesium-137 is the radionuclide of primary concern because of the lichen-caribou-human pathway, although strontium-90 and iodine-131 were of importance also.

   B. Cesium levels were measured by whole body counting. This was done throughout the areas where caribou was a significant food source, and the whole body counting techniques used were sensitive and precise enough to detect cesium body burdens of potential concern.

   C. Strontium-90 levels in humans were assessed from dietary information and measurements in caribou flesh, and to a limited extent from direct measurements in human bone specimens.

   D. Iodine-131 levels were measured in caribou only, but no significant exposure pathway exists for humans.

   E. Other radionuclides including tritium, iron-55, ruthenium-106, cerium-144, plutonium isotopes, and americium-241, have been detected in fish and game, but none were found that appear to be at levels which would pose a hazard to humans.
2. The cancer risk due to the levels of fallout radionuclides in Alaskan Natives is very low, consistant with the observation that the maximum annual dose rates from measured body burdens of cesium were comparable to dose rates from natural background radiation in some regions of the United States (Table V).

Recommendations

1. Individuals on whom cesium measurement data was obtained should be identified so that subsequent cancer development can be determined through matching of individuals in the Alaskan Native Tumor Registry. Cancer occurrence in individuals among this group can then be compared with their measured cesium body burdens, to determine if any correlation exists. The Cancer Branch of the Center for Enviromnental Health, CDC, is willing to work with the Alaskan Native Tumor Registry and the Alaska Department of Health and Human Services in this effort.
2. Aside from the above, no other study of fallout exposure and cancer incidence among Alaskan Natives is recommended at this point. Based on current knowledge regarding radiation carcinogenesis, the radiation doses received and the populations potentially exposed are too small to expect such a study to detect any effect.

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RESOLUTIONS
Adopted by House of Delegates
Alaska State Medical Association
Annual Meeting, June 5-7, 1986

RESOLUTION No. 86-1
SUBJECT: MEDICAID AND GENERAL RELIEF MEDICAL PAYMENTS

WHEREAS, HB-30 was signed into law by Governor Bill Sheffield on February 20, 1986; and

WHEREAS, HB-30 mandates that the State of Alaska pay interest on all undisputed claims that are not paid within thirty days of receipt; and

WHEREAS, HB-30 selectively excluded certain public assistance programs, including Medicaid and General Relief Medical (G.R.M.) from this provision; therefore

BE IT RESOLVED, that the Alaska State Medical Association act in an appropriate manner to effect inclusion of Medicaid payments and General Relief Medical payments in the same manner as any other vendor in the State of Alaska.

RESOLUTION NO. 86-4
SUBJECT: ESTABLISHMENT OF AN ALASKA CHILDREN'S TRUST FUND

BE IT RESOLVED, that the Alaska State Medical Association endorses the establishment of an Alaska Children's Trust Fund for the prevention and treatment of child abuse.

RESOLUTION NO. 86-5
SUBJECT: THANKING THE SPEAKERS

WHEREAS, a highlight of the Annual Meeting is the wide-ranging subject matter presented by our speakers, and

WHEREAS, the speakers have succeeded nobly in stimulating us and have been informative as well, therefore

BE IT RESOLVED, that the Alaska State Medical Association thanks each speaker for his contribution toward making our 41st Annual Meeting a success.

RESOLUTION NO. 86-6
SUBJECT: THANKING THE EXHIBITORS

WHEREAS, the success of the Alaska State Medical Association Convention relies greatly on the support and contributions from exhibiting companies and their representatives; therefore

BE IT RESOLVED, that the Alaska State Medical Association sincerely thanks the pharmaceutical and other companies for their exhibits and contributions toward making our 41st Annual Meeting a success.

RESOLUTION NO. 86-7
SUBJECT: APPRECIATION FOR THE BAR-B-QUE DINNER

BE IT RESOLVED, that the Alaska State Medical Association expresses appreciation to Gary Crabb of the McKinley Village Lodge for his gracious hospitality, the delicious Bar-B-Que, and a fun filled evening that helped make our 41st annual convention a success.

RESOLUTION NO. 86-8
SUBJECT: THANKING THE NATIONAL PARK SERVICE

BE IT RESOLVED, that the Alaska State Medical Association expresses its appreciation to the National Park Service for their assistance and cooperation in making the 41st Annual Meeting of the Alaska State Medical Association a success.

RESOLUTION NO. 86-9
SUBJECT: THANKING THE SALMON BAKE RESTAURANT

BE IT RESOLVED, that the Alaska State Medical Association expresses appreciation to Cliff and Mary Jane Marcum, owners of the McKinley Denali Salmon Bake Restaurant for their gracious hospitality and delightful meal, making the Salmon Bake one of the highlights of the meeting.

RESOLUTION NO. 86-10
SUBJECT: THANKING DENALI RAFT ADVENTURES AND MCKINLEY RAFT CREW

BE IT RESOLVED, that the Alaska State Medical Association expresses appreciation to Gary and Denae Kroll of Denali Raft Adventures and the McKinley Raft Crew for their gracious accommodations in providing an exciting, true Alaskan adventure on the waters of the Nenana River.

RESOLUTION NO. 86-11
SUBJECT: PARTICIPATION IN A COOPERATIVE EFFORT TO REMOVE TOBACCO ADVERTISEMENTS FROM PHYSICIAN AND OTHER HEALTH SERVICE PROVIDER RECEPTION AREAS IN ALASKA

WHEREAS, the American Medical Association declared "unequivocal" opposition to the use of tobacco products and a goal of achieving a "smoke-free"society by the year 2000; and

WHEREAS, the American Medical Association resolved to oppose any form of media advertising of tobacco products (Resolution 114, A-85); and

WHEREAS, efforts by the American Medical Association to convince newspapers and magazines to refuse voluntarily tobacco product advertisements had only modest success (of the 20 most widely circulated magazines in this country, only three do not now accept cigarette advertising); and

WHEREAS, a 1981 Federal Trade Commission report concluded that cigarette advertising may be deceptive because the themes and imagery have a tendency or capacity to deceive by associating tobacco use with good health, youthful vigor, and social and professional success; therefore

BE IT RESOLVED, that the Alaska State Medical Association endorses and will assist in an effort now sponsored by the Alaska Council on Smoking OR Health and endorsed by the Anchorage Medical Society, Alaska Thoracic Society, Alaska Psychological Association, and leading medical authorities in the State and Anchorage Municipal governments, to enlist the cooperation of physicians and other health service providers in Alaska in removing periodicals containing tobacco advertising from physician and other health service provider reception areas, while advising publishers of such magazines that their inclusion of tobacco ads is in conflict with the practice of medicine and health protection.

RESOURCE: Report of the Board of Trustees, AMA,AA (1-85)

RESOLUTION NO. 86-12
SUBJECT: ALASKA NATIVE MEDICAL CENTER (ANMC)

WHEREAS, the ANMC in Anchorage has served the Native community well, first as a TB hospital, then as a general medical center and training hospital, and

WHEREAS, the ANMC in Anchorage is an old hospital that needs extensive and costly repairs to allow it to function as a modern health care center, and

WHEREAS, there already exists an excess of acute care beds in Anchorage in modern facilities, and

WHEREAS, Alaska Natives are provided health care by private facilities in many communities including Fairbanks, Juneau, and Ketchikan, and

WHEREAS, many Alaskan Natives are forced to leave their community hospitals and to go to Anchorage to the ANMC for evaluation and treatment when they would prefer to stay in their own community hospital, and

WHEREAS, many Alaska Natives receive health care coverage from a variety of programs, and more each year seek health care through private facilities, and

WHEREAS, the cost of modern hospital care has become so expensive that society cannot afford long hospital stays and excessive acute care beds, therefore

BE IT RESOLVED, that the Alaska State Medical Association encourages the Indian Health Service **not** to build another Native Hospital in Anchorage, but rather to provide health care through modern facilities already available in the community, and

BE IT FURTHER RESOLVED, that the Alaska State Medical Association encourages the Indian Health Service to build a facility in Anchorage similar to Providence House where Natives and possibly non-natives from rural Alaska can stay at a reasonable cost while they are receiving outpatient evaluation and treatment.

RESOLUTION NO. 86-13
SUBJECT: CANCER RESEARCH CENTER PLANNING GRANT

WHEREAS, cancer is one of the leading causes of death in Alaska; and

WHEREAS, there have been documented excess incidences of certain types of cancer among Native and non-native Alaskans; and

WHEREAS, in 1983 the Alaska State Medical Association supported the "Feasibility Study to Determine the Need for a Cancer Research Center in Alaska"; and

WHEREAS, in 1984 the Alaska State Legislature, Governor, and University of Alaska set aside

$35,000 to fund this feasibilty study; and

WHEREAS, in July 1985 the feasibility study was completed and determined that there is a definite need for a cancer research center in Alaska with a broad base of support among the Alaska State Medical Association, Alaska Dental Association, Alaska State Nurses Association, American Cancer Society, native corporations, hospitals, and innumerable other societies and organizations; therefore

BE IT RESOLVED, that the Alaska State Medical Association recommends that the Governor, Legislature, and Department of Health & Social Services allocate $40,000 to continue the planning grant requested by the University of Alaska in 1985 for developing a Cancer Research Center in Alaska.

RESOLUTION NO. 86-15
SUBJECT: HEALTH STATISTICS

WHEREAS, the Alaska State Medical Association has repeatedly called for responsible action by State government regarding health statistics collection and tabulation; and

WHEREAS, the administration's response has been to cut positions rather than to add them and thus render the problem worse rather than better; therefore

BE IT RESOLVED, that the Alaska State Medical Association again urges the Governor and Legislature to fund appropriately, staff adequately, and collect and tabulate responsibly, health statistics for the State of Alaska.

RESOLUTION NO. 86-16
SUBJECT: STATE MEDICAL BOARD

WHEREAS, the Legislature and Governor have again funded and staffed the Alaska State Medical Board at a woefully inadequate level, and

WHEREAS, those who choose to be critical of the Alaska State Medical Board most often cite inadequacies of investigation directly related to insufficiently skilled, experienced and supervised staff; and

WHEREAS, physician licensing fees were raised by the administration with the stated purpose of appropriately supporting the Alaska State Medical Board, yet this has not occurred; therefore

BE IT RESOLVED, that the Alaska State Medical Association call upon the Governor and Legislature to fund the Alaska State Medical Board at a level at least sufficient to maintain a full-time professional and secretarial staff and to hold at least the statutorily required number of meetings each year.

RESOLUTION NO. 86-17
SUBJECT: WAMI PROGRAM AND MEDICAL EDUCATION IN ALASKA

WHEREAS, the WAMI program was established in 1971 in order to provide access to medical education not available through the existing WICHE Program, to encourage graduates to enter primary care, and to help correct the mal-distribution of physicians; and

WHEREAS, in the subsequent 15 years, of the 42 graduates of the WAMI program that have finished medical school and residency programs and are now in practice, 23 have returned to Alaska and an additional 23 non-Alaskan WAMI graduates have also elected to practice in this state, proving that the WAMI program is successfully meeting its first goal; and

WHEREAS, WAMI graduates are selecting primary care at a rate of three times the national average, proving that the program has been successful in meeting its second goal; and

WHEREAS, WAMI graduates are now practicing in eight rural Alaskan communities, and 15 WAMI trained physicians are in hospitals and clinics primarily servicing Native Alaskans; and

WHEREAS, other unanticipated benefits of the WAMI program have accrued over these fifteen years, including:

> Medcon telephone consultation,
> Continuing medical education by visiting University of Washington Medical School faculty,
> Third year medical student clinical rotations in OBGYN, family medicine and psychiatry,
> Itinerant birth defect clinics,
> Psychiatric residency rotations, and
> MEDEX out-reach program, recruiting physician assistant students for practice in bush communities; and

WHEREAS, to this date no alternate, more cost-effective program that meets all of these goals has been developed; and

WHEREAS, the University of Alaska, Fairbanks recommended termination of involvement in the WAMI program with no students to be admitted after September, 1986; and

WHEREAS, no attempt was made to study the impact of this precipitous, ill-advised and mis-informed decision or to solicit input from Alaskans who may suffer adverse impacts as a result of the potentially catastrophic consequences to Alaska's medical care system; and

WHEREAS, this proposal appears to be entirely in opposition to recent recommendations to expand arctic health research activities of the American Medical Association, Alaska Public Health Association, American Society for Circumpolar Health, Anchorage Medical Society, American Public Health Association, American Association of Public Health Physicians, Polar Research Board of the National Academy of Sciences, U.S. Arctic Research Commission, University of Alaska Foundation Scientific Advisory Commission, and Legislature of the State of Alaska; and

WHEREAS, the WAMI Program has achieved unique success in attracting Alaskans to the medical professions, in assuring that Alaskans have the opportunity to be accepted into medical school, and in improving the distribution of highly trained physicians and other health care providers in practice throughout Alaska; therefore

BE IT RESOLVED, that the Alaska State Medical Association urges the President and Board of Regents of the University of Alaska to overturn the administrative decision to eliminate the WAMI Program, to reaffirm their commitment to the WAMI Program, and to review independently the WAMI Program, taking into account its past performance and its potential to play a crucial role in implementing the goals of Congress as stated in the National Arctic Research & Policy Act of 1984.

RESOLUTION NO. 86-18

SUBJECT: PROPOSED REVISION OF STATUTES RELATIVE TO MEDICAL MALPRACTICE

WHEREAS, best information available to ASMA as of this date suggests that the Alaska Court System is presently involved in a proposed revision of Alaska Statutes relating to medical malpractice in the State of Alaska; and

WHEREAS, such revision clearly affects all physicians and therefore their patients, and all citizens of the State of Alaska; and

WHEREAS, there are presently significant numbers of physicians who are unable to afford liability insurance, thereby leaving large numbers of citizens in the State of Alaska without protection in the event of injury sustained while seeking medical care; therefore

BE IT RESOLVED, that the Alaska State Medical Association respectfully requests the Alaska Court System to seek the participation of physicians of the Alaska State Medical Association regarding such proposed revisions to the medical malpractice statutes.

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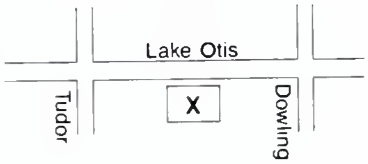

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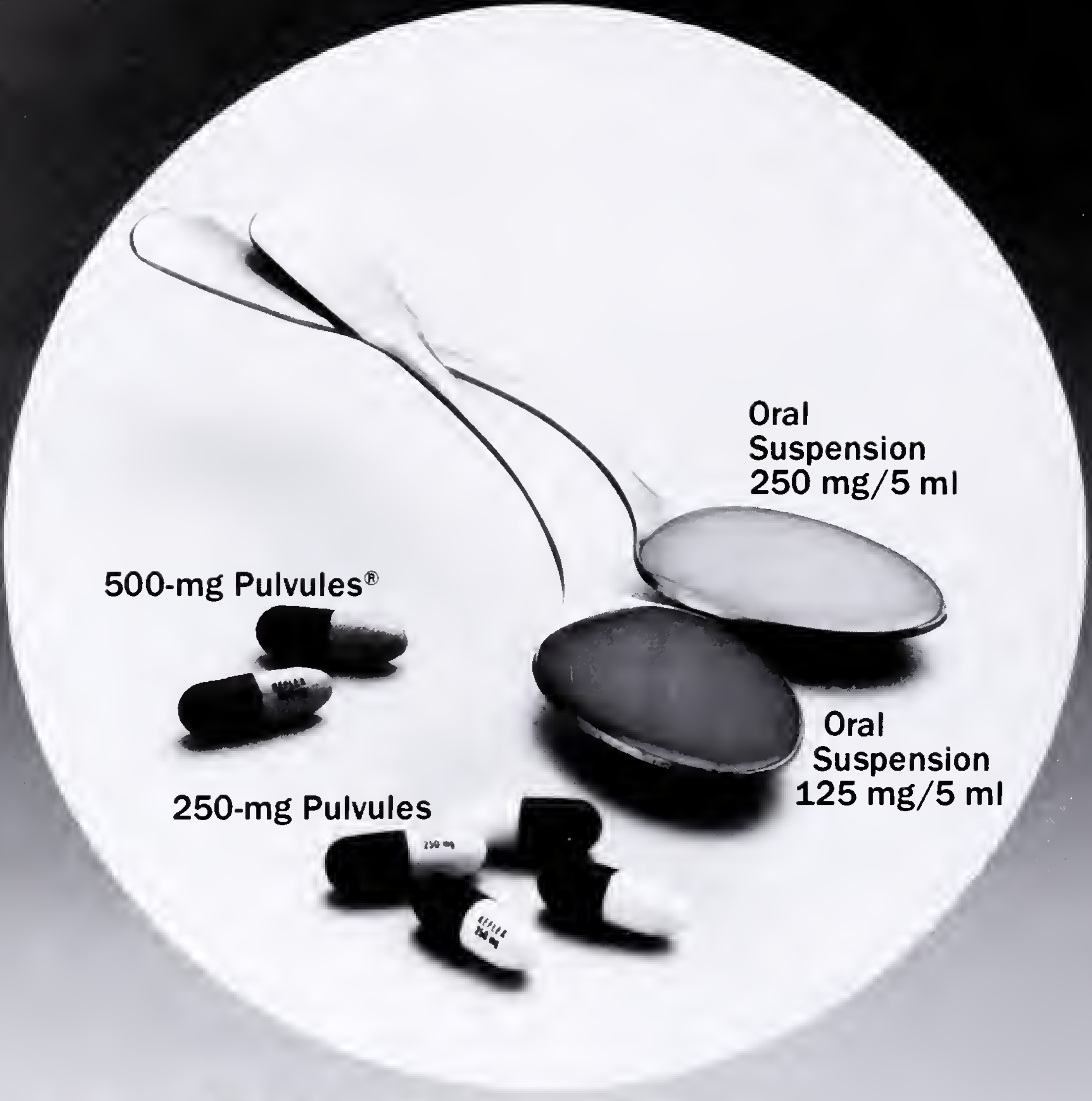

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REPORT TO THE AMERICAN MEDICAL ASSOCIATION UNITED STATES ARCTIC RESEARCH POLICY

JUNE 12, 1986

By the Alaska State Medical Association

The American Medical Association played an important role in developing national policy with respect to arctic research. Tremendous progress has been made since June 1983 when the AMA House of Delegates passed a Resolution pertaining to "Research on Physical and Mental Health Under Arctic Conditions."

This AMA resolution, jointly submitted by Dr. Ed Press, Delegate from the American Association of Public Health Physicians and Dr. Richard Witt, Delegate from the Alaska State Medical Association, played an integral part of a national effort by physicians and other health care organizations and agencies to develop a viable and effective national arctic health science policy. By passing this resolution, the AMA joined the American Public Health Association, which passed a similar resolution at its annual meeting in November of 1982, in this national effort. Subsequently, numerous other national and local medical and public health organizations endorsed the concepts in the AMA resolution and worked with the AMA to amend pending congressional legislation to provide for a "...comprehensive national policy with respect to arctic research". At the time when the House of Delegates acted, health research was not included as a component of the proposed congressional legislation to establish a national policy with respect to arctic research.

The AMA efforts were effective in having the proposed bill amended. The Arctic Research and Policy Act of 1984 was signed into law by President Reagan in August 1984. Health is a prominent and an integral component of the bill. Since passage of the bill, major progress has been made in implementing the concepts contained in the AMA resolution.

As required by provisions of the bill, an Interagency Arctic Research Policy Committee and a United States Arctic Research Commission were appointed by the President, and the National Academy of Sciences developed a 5-year plan to implement the National Research Policy.

The World Health Organization, Regional Office for Europe, established working groups to provide guidance on arctic health research, the 6th International Symposium on Circumpolar Health was held in Anchorage, Alaska in May 1984 with 770 participants from 22 countries, and last year the Proceedings of this Symposium were published. In August 1984, the American Public Health Association published the **National Arctic Health Science Policy** developed by the American Public Health Association Task Force, in its APHA Report Series.

In March 1986 the International Union for Circumpolar Health was founded by representatives of the Soviet Union, Nordic Council for Arctic Medical Research representing the Scandinavian countries, Canadian Society for Circumpolar Health, and American Society for Circumpolar Health. We expect the Scientific Committee for Antarctic Research and the World Health Organization also to participate in the future.

Only a few weeks ago, the Alaska State legislature passed legislation to establish an Alaska Research Policy and a Science and Engineering Advisory Commission.

The actions of the AMA House of Delegates in June 1983 have resulted in exciting developments that will improve our nation's ability to conduct arctic health research.

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UNITY

James E. Davis, M.D.
Speaker of the House of Delegates
American Medical Association

In June, I had the very great pleasure of attending the Alaska State Medical Association's 41st Annual Meeting, in Denali National Park. The trip was unlike anything I've experienced. I came with a sense of adventure and a curiosity about Alaska, a desire to see the land and come to know some of the members of ASMA. I was not disappointed.

The warm welcome I received, the natural beauty of the landscape, and the experience of flying over snow-capped peaks, very close to the great Mount McKinley, was an experience I'll never forget.

It was difficult, at the time, not to lose sight of the fact that I was in Alaska to discuss a very serious and important subject: unified membership with the AMA. And I am very pleased to have this opportunity to again address the subject of unified medicine for the readership of **Alaska Medicine.**

I cannot recall, either from personal involvement or from reading the history of medicine, a time when the medical profession has faced the crisis it faces now—government control, third party tyranny, malpractice suits, loss of prestige. If ever there were a time when physicians need to be unified and pull together, it is now.

The matter of unified membership, coming as it does at this time of crisis, may cause some physicians to lose sight of the critical issues that face all of us. However, it is precisely these crises that precipitate the necessity to strengthen medicine's unity. We need to be stronger and support all of the organizations that help to protect our profession. If we lose our professionalism, both we and our patients will suffer.

A well-known labor lawyer made this observation: "Physicians must give up a little of their individual freedom today if they are to preserve for tomorrow their greater freedom to practice medicine as independent professionals. If they don't, and if current trends continue, the physician will be, in fact, an employee as determined by the formal definition of labor law, and he or she will have no effective negotiation option except to unionize."

I urge all physicians in the State of Alaska to become, or remain, members of all levels of organized medicine: your local societies, the Alaska State Medical Association, specialty societies, and the American Medical Association. I urge all ASMA members to support the bylaws amendment that was introduced at your Annual Meeting in June, calling for unified membership with the AMA.

Once unified, all members will receive a 10% discount on AMA dues and access to a broad and growing range of information services. Unified societies will be provided with increased AMA services and staff support for such things as special projects, AMA briefings on important issues for medical society officers and executives, and participation in the Unified Societies Advisory Committee consisting of representatives of all unified societies. These and many other AMA benefits to unified societies and their members are important and well worth the payment of AMA dues in their own right.

But what unification really means is that our entire profession will be stronger. Each level of our federation has its own primary focus and concerns at the local level, the medical specialty level and, through the AMA, at the national level. More often than not our concerns are shared, and when we make unified responses, our presence in social, economic and political (as well as clinical) forums is that much more powerful.

I hope to visit Alaska again, to once again experience its beauty, and renew and extend medical friendships make there. But before I do, I hope you will come to the conclusion that unification will strengthen all of our organizations and help them to represent us more effectively. It will allow us to gain more control over our profession and our futures. That is why I encourage you to support unified membership.

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AMS AUXILIARY PRESIDENT'S MESSAGE

Ideas are being formulated for the 1986-1987 Anchorage Auxiliary. The board has met, and the action is beginning.

Pat Durham, the American Medical Association National President, will visit Anchorage for the "Fall Salad Luncheon" on September 3. Speaking with her by telephone, she is very excited about her trip to Alaska. Mrs. Durham's goal this year is to remind members that they belong to a very valuable organization with a proud tradition. The auxiliary is medicine's number one ally and is an influential organization. Another of her major goals is to promote more cooperation between the auxiliaries and their medical societies.

October holds plans for a "Totally Tacky and Tasteless Trot" party, which should be real fun. Tickets will be appropriately cheap, entertainment exciting in it's own special way, and food and drink of questionable quality. Keep your eyes open for this one.

November will be devoted to community service in cooperation with other organizations. We are also planning a children's health fair in the spring.

With elections this fall, the auxiliary will be involved in informing ourselves and others of the candidates views on tort reform and other medical related issues.

The Anchorage Medical Society Auxiliary is a group of interesting, informed, community and health oriented people. We would like to see the membership grow in order to have greater energy and ability to accomplish our goals.

Alice Samuelson

ASMA AUXILIARY PRESIDENT'S MESSAGE

My principal message is that your State Auxiliary plans to be right there alongside you, working with you to fine-tune tort reform and to try to fill a great need in Alaska. The latter is the financial sponsorship of Champ Camp. For those who are not familiar with it, Champ Camp was begun three years ago by the Alaska Lung Association and is medically directed by pediatrician Thad Woodard, a man with a personal interest in and commitment to dealing with asthmatic patients. The financial backing the camp had was withdrawn by it's primary source, and we are going to do all we can to fill in the gaps this year, while the Lung Association searches for another contributor. Keep in mind that asthma covers nearly half of all pediatric chronic illness, and over 2,700 Alaskan children must deal with a moderate-to-severe level of it. So many of these little ones lead limited lives with frequent hospitalizations, that nationally the American Lung Association decided to design a summer camp focus for these kids who are taught about the disease, causes, how to administer their own medications, and exercises they can handle without wheezing. Camp provides one of the few times in their year when they do not feel "different," since they all have asthma. The self-esteem and self-discipline they develop is marvelous to behold, and these growths carry over into other aspects of their lives. Taking responsibility for their illness/wellness, medications, and attitude affects personal relationships, school, play, and continuing maturity. It's a tall order for these children who come to Camp Peggy Lake from Fairbanks, Kodiak, Talkeetna, Palmer, Juneau, Anchorage and Wasilla. We in your auxiliary hope that you are as excited about sponsoring Champ Camp as we are! Looking forward to a great year, I am.

Patrice Gerster

PRESIDENT'S PAGE

Greetings to Alaska State Medical Association members and others who read these pages, from your new ASMA President. I look forward to the opportunities of the coming year and to working with many of you.

I have just returned, along with Dave Johnson and Doug Smith, from the Annual American Medical Association House of Delegates Meeting in Chicago. A major topic was the passage of a proposal for the financing of Medicare into the next century, to correct the present deficit problem and build a trust fund that will prevent the repetition of Medicare's current problems. Associated with this is the idea of health IRA's, with the thought that long-term care could be provided for in this fashion since it would be very difficult to finance long-term care through tax money. These proposals have been given a great deal of thought by the AMA for almost two years now before their passage on the floor this June. They have great merit and will now be brought to Congress by the AMA with the hope of obtaining legislation along these lines in the near future.

The AMA also endorsed the restriction of tobacco sales to individuals at least 21 years old, the inclusion of warnings on alcoholic beverage containers regarding health affects and is considering working with advertisers to eliminate alcohol advertising whose main appeal is to youth. Many other issues were presented and dealt with. I was strongly impressed by the quality of both committee work and House of Delegates deliberations. Amid all the "busy-ness" and the multiplicity of issues, it is amazing how much common sense comes out of these deliberations.

We of the ASMA are going to be dealing with the issue of unified membership with the AMA over the coming year. As you may know, a motion is on the table from our June 1986 meeting to amend our bylaws to provide for unification, which would require each ASMA member to also be a member of the American Medical Association. I believe this has great merit, and members will be receiving a letter in the near future from David Johnson, Doug Smith, and me regarding this issue with a request for feedback. Please look for this in your mail and provide us with your views. It is my view that membership in the AMA is fully as important to each of us as membership in the Alaska State Medical Association, as there are major issues of national health policy being decided now that will affect all of us for many years to come. We need to strongly support the one organization that speaks for all of us, which has done excellent work in our behalf in recent years.

We are moving into new quarters, across the street from our old building, for at least the next few years, because of the circumstances of the real estate market in Anchorage and our present tenant's need for increased space. Your executive committee and council feels that this is a wise and prudent move, and I believe you will find that our new quarters are attractive and very accessible.

I look forward to working with all of you and with our new executive director and expect to have a busy and pleasurable year.

Don Thieman, M.D.
President

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BOOK REVIEW

Circumpolar Health '84
Proceedings of the
Sixth International Symposium

Edited by Robert Fortuine, M.D.

Publication date: April 16, 1986

Accelerating exploration and resource development in high latitudes of both the northern and southern hemispheres has been accompanied by substantial changes in all aspects of the the health challenges that are inherent and unique to these regions. Physiological, medical, social, cultural, and historical aspects of circumpolar health interact to such a degree that the international scholarly community founded the Symposium on Circumpolar Health in 1967, and it has convened every three years since then to facilitate the interdisciplinary awareness of changes in health-related fields.

This volume is the synthesis of the 1984 symposium that took place in Anchorage, Alaska, where participants from 22 countries were exposed to a wide range of subjects. Over 100 articles are included here, grouped into these major sections: general surveys; physiology and pathology of cold climates; demography, morbidity, and mortality; infectious diseases; non-infectious and chronic diseases; acculturation, mental health, and substance abuse; health programs and manpower; and progress in self-determination.

Circumpolar Health '84 will be a useful resource for a wide range of readers including physicians, physiologists, policy-makers, and sociologists.

Robert Fortuine was the director of the Public Health Service Alaska Native Medical Center, Anchorage, where he is now the deputy chief of the department of family medicine and maintains a full-time clinical practice. He was with the Indian Health Service for 21 years and, from 1977 to 1980, served as the International Health Attache at the U.S. mission to the United Nations in Geneva.

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3.	23:43	Kim Smith, M.D.
4.	24:23	Kirk Coles
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15.	26:08	Bob Kerr
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21.	27:57	Gayle Thieman
22.	28:00	Kathleen Todd, M.D.
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November 13-15, 1986

Anchorage, Alaska: The Eleventh Annual Alaska Symposium on Emergency Medical Services will offer a variety of educational programs. These workshops will provide continuing medical education credits applicable toward EMT and paramedic recertification requirements, CEARP points for nurses, and Category I CME credits for physicians and physician assistants. Contact: Charles Ramage, Associate Coordinator, EMS Section, Division of Public Health, Department of Health and Social Services, P.O. Box H-06C, Juneau, Alaska 99811, (907) 465-3027.

Announcement

The Annual Meeting of the United States-Canadian Division of the International Academy of Pathology will be held at the Palmer House in Chicago, Illinois starting with "Companion Meeting" symposia on Sunday, March 8 and continuing through Friday evening, March 13, 1987.

Scientific Papers, Poster Sessions, thirteen Specialty Conferences, and 44 Short Courses are scheduled. Two Special Courses will be offered on "Diagnostic Cellular and Molecular Pathology" with Dr. Benjamin Trump as Course Director and "Advances in the Application of Immunoctyochemistry to Diagnostic Surgical Pathology" with Dr. Cecilia Fenoglio-Preiser as Course Director. The Long Course will be on "Pathobiology and Recognition of Malignant Melanoma" with Drs. Martin Mihm and George Murphy as Course Directors.

The Maude Abbott Lecture will be delivered by Dr. Emmanuel Farber on Tuesday, March 10.

Timely Topics in Pathology will feature a session on "Telepathology - Delivery of Pathology Diagnostic Services in the Future" presented by Dr. Ronald S. Weinstein.

A special lecture entitled "The Changing Faces of Pathology and Medicine" will be given by Dr. Peter Ward.

Further information about the meeting and courses may be obtained from Dr. Nathan Kaufman, Secretary-Treasurer, United States—Canadian Division of the International Academy of Pathology, Building C, Suite B, 3515 Wheeler Road, Augusta, GA 30909. Telephone (404) 733-7550.

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EDITORIAL

Internationally known health economist, Alain Enthoven, eloquently catalogued problems facing physicians in private medical practice in his presentations at the annual meeting of the Alaska State Medical Association at Mt. McKinley Park in June. In fact, Dr. Enthoven's sobering message was that sweeping changes wrought by economic forces shaped by the federal government and the private sector will reshape medicine and make traditional private medical practice a thing of the past.

According to the scenario painted by Dr. Enthoven, no longer will physicians be able to adhere to Osler's affirmation that, "The practice of medicine is an art, not a trade; a calling, not a business." Rather, the practice of medicine will succumb to market forces and be thrust into the mainstream of competition, business, and free-enterprise. Health services in the future will be delivered by the low bidder—all in the name of cost-containment.

There clearly is no place in this future for a William Osler. There is clearly no place in America for this aberration of the practice of medicine.

The message of the federal government and, increasingly, of private employers—that health care costs are too high and must be restrained—has been widely heard but poorly understood. A superficial solution is evolving—restrain excessive costs by further restraining physician's fees and by promoting competition. This message can only bring to mind a comment of an ancient sage, Cicero, "When you have no basis for an argument, abuse the plaintiff."

Recently, in **Issues in Science and Technology,** former Health and Human Services Secretary Joseph Califano, Jr., wrote of the experience of Chrysler Corporation in restraining health costs in Chrysler's employee health program. Mr. Califano, currently a director of Chrysler Corporation, described Chrysler's efforts to reduce expenditures through competitive bidding of services and hard business negotiations. He made particular note of reducing payments for physician services. Yet, figures he quoted in his article showed that 93% of the $58 million that Chrysler saved came from reductions in payments for laboratory and hospital services and charges, not from reductions in physician's fees.

Where are the costs occurring? Where are charges being made? "Charges may bear little resemblance to economic cost, and use of charges as a proxy for economic cost may lead researchers to draw unwarranted conclusions about economic efficiency." (1)

Even a cursory look at medical practice today cannot help but see the vast health care industry that has multiplied about patients and their physicians. Combine the laboratory industry, pharmaceutical industry, hospital supply industry, nursing home industry, insurance industry, and the administrative industry and you have a ready explanation for where our health care dollars are going. Yet, why are these industries escaping scrutiny? Why are they apparently insulated from competitive market forces? Why are we being led to believe that physicians are at the root of "runaway health costs"?

As Lewis Carroll so eloquently stated "If you don't know where you are going, then it doesn't matter which road you take to get there." If we don't ask the right questions, we won't get the right answers. Why do patients get charged such high amounts for laboratory tests that take only a few minutes to run and cost only a few pennies? Why do patients get charged excessive sums for hospital supplies such as pre-packaged, disposable suture removal kits or lumbar puncture kits that cost only a few dollars to make? Why are patients charged many times the costs for laboratory diagnostic tests—herpes or chlamydia cultures, for example? And why are the costs of administrative requirements of government and private sector programs never subjected to the same stringent cost-benefit and cost-effective analysis that are demanded for patient services and procedures?

Instead of carefully analyzing all segments of the health service industry, government has selectively and prejudicially directed its attention at physicians. To the degree that abuses have been enumerated and corrective actions implemented, such efforts are to be applauded. But, all studies have documented the relatively small proportion of the total health dollar that goes to physician fees. It is past time for the government, private industry, health economists, and organized medicine to scrutinize the rest of the health care industry.

Absent in this new business of medicine is any assurance of quality of medical care. Economists and health planners tinker at will with the medical care system. While they experiment with financing mechanisms in their attempts to curb costs, they pay only lip service to quality of care issues. We must preserve the art of medicine., the calling of medicine, and the practice of medicine. We must remain advocates of our patients. Those in need of healing, those who are suffering, those who are ill and in need of care are not our clients. They are our patients. Our duty lies with them.

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[1] Finkler, Steven A, The distinction between cost and charges, Ann Intern Med. 1982;96:102-9.

LETTERS TO THE EDITOR

Dear Editor:

This letter is a clarification of the discussion appearing in the article **Cost Savings of Regionalized Perinatal Care For Low Birthweight Infants: Part I. Mortality,** written by me in the April/May/June 1986 issue of Alaska Medicine. Although the article dealt with many issues relating to progress in regionalization of perinatal care the section of the article that raised the greatest controversy was related to the participation of the physicians practicing obstetrics at Humana Hospital-Alaska in high risk maternal transport.

I regret any implication in the discussion that Humana Hospital-Alaska or its administration played any part in the failure to transfer appropriate high-risk mothers to the tertiary care center at Providence Hospital. In fact, Humana Corp. did not own the present facility for a portion of the time covered in the data. Indeed, the administration of Humana Hospital-Alaska has supported the concept of regionalization by committing a significant amount of money and manpower in upgrading their perinatal services at a Level II status. They have participated in the publication of the perinatal transport manual (1) for Alaska, developed a 24 hr., in-house anesthesia service that has resulted in the ability to do an emergency cesarean section quicker than at the tertiary care center, and improved physician presence at certain high risk deliveries (such as meconium staining) resulting in an overall improvement in perinatal outcome for infants delivering at Humana Hospital.

Medical practice in Anchorage is such that physicians clearly determine where their patients are hospitilized. I feel that Anchorage is somewhat unique in that physicians practicing obstetrics have such a strong preference for one or another hospital that they generally restrict their practice to the hospital of their choice. In fact, this may be the primary reason for the difficulties with maternal transport discussed in the article.

Sincerely,
Jack Jacob, M.D.

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1. Jacob J, Stilson M, Hanson H, Gronau S: Stabilization and Transport of the High Risk Mother and Neonate. Published by Northern Printing Inc., Anchorage, Alaska, 1986.

Dear Editor:

The convention is over and really seemed to be enjoyed by everyone. The Auxiliary is most appreciative of the generous $500 donation given to the Auxiliary by the ASMA to help pay the expenses of our speaker, Steven Debore. It certainly helped defray our costs, and we believe Mr. Debore added an interesting dimension to the convention.

The year has been a successful one for the auxiliary. Total membership was up, and at the close of our fiscal year, 140 members had pined through to the national level. This was a second high for state Auxiliary.

As always the auxiliary is willing to help in any way in the coming year.

Thank you for your support
Sincerely,
Carolyn Crouch
President 1985-86

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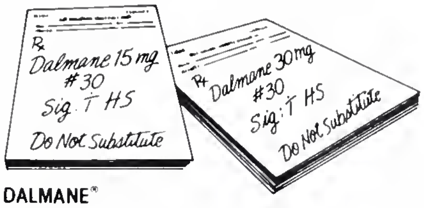

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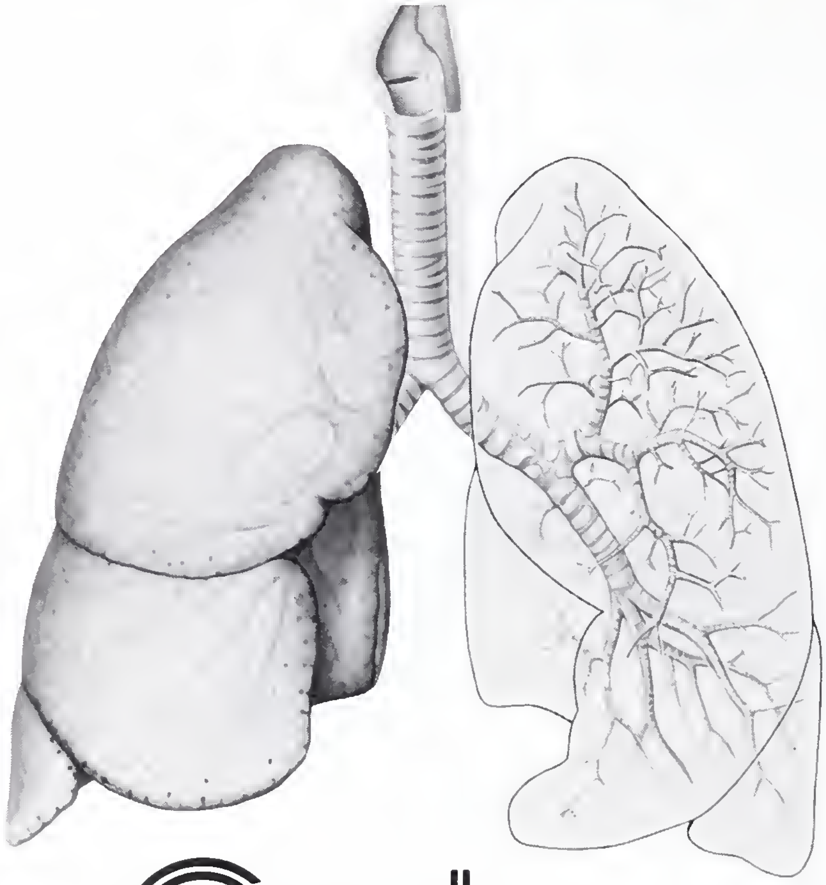

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Arthritic Findings in the Hands of Alaska Natives with Rheumatic Heart Disease—with Presentation of a Case of Post-Rheumatic Fever Arthropathy

by David W. Templin, M.D.[1]

ABSTRACT

Eighty-five Alaska Natives with rheumatic heart disease were examined for the presence of arthritic findings in the hands. Two had rheumatoid arthritis (RA), seventeen had degenerative joint disease (DJD), one had mixed connective tissue disease (MCTD), one had Reiter's syndrome, and four had other non-arthritic changes. One case of Jaccoud's arthropathy was found and described. The prevalence of arthritic findings in the hands of the 85 patients is consistent with that expected from population studies.

INTRODUCTION

Although acute rheumatic fever (ARF) and rheumatoid arthritis (RA) are clearly delineated as independent disease entities, the occurrence of a peculiar arthritis associated with chronic rheumatic fever (and occasionally systemic lupus erythematosus) has been reported repeatedly since Jaccoud's description of this syndrome (1–17). The accumulation of case reports and reviews suggests that there is, indeed, a form of arthritis which involves primarily the hands and occurs in patients who have had severe or recurrent ARF. Those patients with chronic post-rheumatic fever arthritis (CPFA) or Jaccoud's polyarthritis are, with few exceptions (5), seronegative and have no evidence of synovitis.

Most patients with Jaccoud's polyarthritis have a history of a severe attack of rheumatic fever with a prolonged course or with repetitive episodes and have progressive deformities of their joints with subsequent attacks. The deformity, which involves the hands and the feet, is predominantly fibrosis of the periarticular tissues with flexion deformities of the metacarpal-phalangeal (MCP) joints, soft tissue swelling, tendon crepitus and ulnar deviation, most marked in the 4th and 5th MCP's. Abnormalities of the joint surface are generally absent. Hyperextension of the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints has been noted; and there is an inconstant finding of an erosive change of the metacarpal head on the palmar and radial aspect, forming a hook-like appearance on the radiograph (12).

For a number of years it has been recognized that there is a very high incidence of ARF and rheumatic valvulitis among the Alaska Natives (18–24). In some areas inhabited by Eskimos, the reported annual rate of ARF has continued to be 50 to 60 per 100,000 (22–24). Approximately 400 patients with diagnosed rheumatic valvulitis have been identified and listed on the chronic disease register and 6–7 patients undergo valve surgery annually. Until recently, fresh cases of ARF had been seen yearly by the physicians of the Alaska Native Health Service (24). The reason for the high incidence of rheumatic heart disease is not known.

Studies by the Center for Disease Control extending over the past 15 years have shown that in certain Eskimo schools the carrier rate for pathogenic beta-hemolytic streptococcus was as high as 30% (18–20). In addition to asymptomatic carriers, there was a high rate of symptomatic streptococcal pharyngitis. A control program, similar to one in Wyoming, was carried out in selected villages (20, 24). Although patients with rheumatic heart disease (RHD) live in all parts of Alaska, the greatest number are found living along the lower portion of the Yukon and Kuskokwim Rivers and in the delta area between these rivers (22).

It appeared that this would be a satisfactory group to evaluate for the occurrence of chronic post-rheumatic fever arthritis and other rheumatic conditions which might demonstrate some relation to RHD.

METHODS

A prospective study evaluating the rheumatologic findings in the hands of patients with known valvular disease was begun March 15, 1978. The patients were those Alaska Natives presenting themselves at the Alaska Native Health Service Hospitals and Clinics, either for primary care or referral, specifically for consultation in internal medicine or cardiology clinics. All patients had been evaluated in a cardiology clinic

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[1] Chief of Medicine, Alaska Native Medical Center, Box 7-741, Anchorage, AK 99510

and the diagnosis of RHD established by a cardiologist. Many of the patients had undergone cardiac catheterization or echocardiographic studies.

The author examined the hands of all the patients included in the study. Because the arthropathy of the CPFA is seen predominantly in the hands and feet, it was decided to limit the required examinations to the hands. The feet and other joints were not examined in most cases due to logistic difficulties arising from the clothing necessary in the land of cold temperatures.

In patients with a history of physical findings of arthritis in the hands and wrists, a radiograph of the hands was obtained and a complete joint examination was performed. Rheumatoid factor determination was performed on those with arthritic findings other than degenerative joint disease (DJD). All patients were questioned about current or prior joint pain, morning stiffness, swelling, heat, and loss of motion in all joints as well as in the hands and about episodes of ARF, and their medical records were reviewed in regard to this information. Records prior to 1960 were available for only a few patients.

Patients who had aortic valve disease—along with no history of ARF—or who had clinical findings suggesting calcific aortic stenosis or congenital aortic valve disease were excluded from the study. The few patients with a history of ARF and findings suggesting prolapsing mitral valve only were also excluded.

RESULTS

Eighty-five patients, 33 men and 52 women, satisfying the criteria for the study, were examined during the 12 months. The ages ranged from 14 to 78 years (mean 40.2). Fifty-nine gave a history and had indication in their medical records of an episode compatible with ARF. Only six had suffered clinically evident, repeated episodes of ARF. Four patients had isolated involvement of the aortic valve, and 30 had isolated disease of the mitral valve. Twenty patients had undergone cardiac surgery, including eight who had a prosthetic mitral valve replacement, four who received prosthetic aortic valves, and one with replacement of both aortic and mitral valves. Six patients had mitral commissurotomies alone, and one had a mitral annuloplasty.

Fifty-four patients had no abnormalities of the hands other than the mild hyperextension of the MCP noted in our patients. Another nine had hand abnormalities due to trauma, frost bite, hemiparesis, or amyotrophic lateral sclerosis. In 17 (10 women and 7 men) clinical findings of DJD of the hands were noted. Their average age was 55.5 years. One man had arthralgias of the left hip, left knee, and right second PIP joint without synovitis or loss of motion on examination. He had chronic back pain and radiographic evidence of sacroiliitis. There were no ocular or genitourinary symptoms or findings. The HLA-B27 was positive.

Two patients had rheumatoid arthritis; one classic and the other definite by American Rheumatism Association (ARA) criteria. Both were seropositive.

One patient had Raynaud's phenomenon, sclerodactyly, arthralgias, and radiographic evidence of esophageal and small bowel motility abnormalities. The anti-ribonuceloprotein titre was 1:100,000, satisfying the diagnosis of mixed connective tissue disease.

Another patient, whose case is described in greater detail, had the clinical findings of Jaccoud's arthritis.

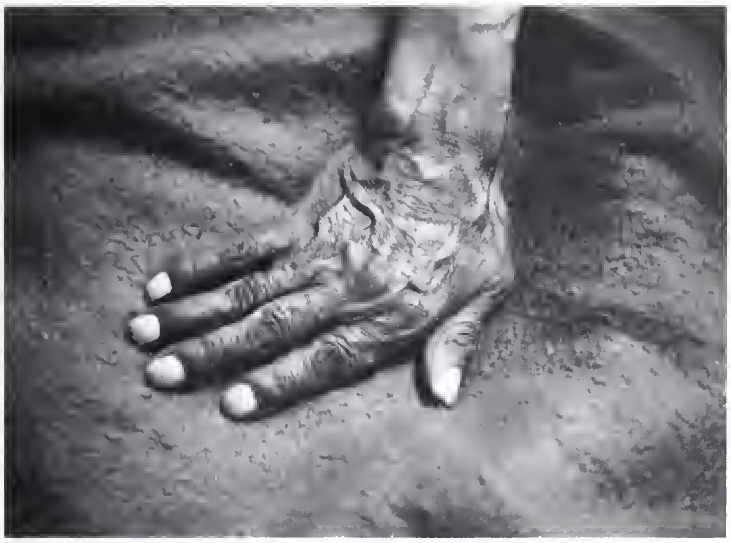
CASE REPORT

A seventy-seven year old blind, nearly deaf, Eskimo man residing in a nursing home was referred for evaluation of his cardiac pacemaker and rheumatic heart disease. Historical details are limited because of his blindness and deafness. A review of his medical records indicated that during the 1940's he had an episode of ARF and subsequently had progressively developed the murmurs of aortic stenosis, aortic insufficiency, and mitral insufficiency. He was asymptomatic until 1975 when he developed complete heart block. At that time a permanent transvenous pacemaker was implanted. Subsequently, mild cardiac decompensation required treatment with digoxin and hydrochlorothiazide.

The first indication of arthritic complaints was in 1961 following a fractured wrist. In 1965 a diagnosis of rheumatoid arthritis was made. He denied any joint pains, although deformities of his hands and feet were noted. In 1974 he was evaluated for a painful hallux valgus deformity and hammer toes of the 2nd, 3rd, and 4th toes on the left foot.

On examination, he was an elderly, nearly deaf man who understood Eskimo only. He had dense cataracts. There were a few basilar rales and hepatojugular reflux. The PMI was displaced laterally. The murmurs of aortic stenosis, mitral regurgitation, and aortic insufficiency were present. There was marked ulnar deviation of the right MCP joints, with no fixed deformity, which could be returned to a normal position voluntarily or passively. Hyperextension of the second, third, fourth and fifth PIP joints was present, producing the appearance of mild "swan neck" deformity. All of the deformities were completely reducible passively and voluntarily. A full range of motion was present with no swelling, tenderness, or pain. The extensor tendons slipped laterally during passive flexion in ulnar deviation to lie in the troughs between the MCP heads (Figures 1-4). A deformity of the right knee appeared to be bony swelling and instability of the medial and lateral ligaments and the cruciate ligaments.

There was no evidence of fluid and there was a full range of motion without pain. Bilateral hallux valgus



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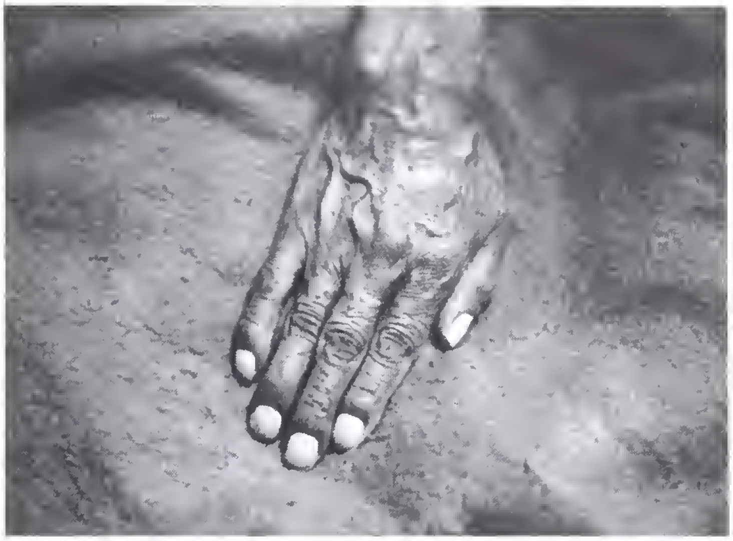

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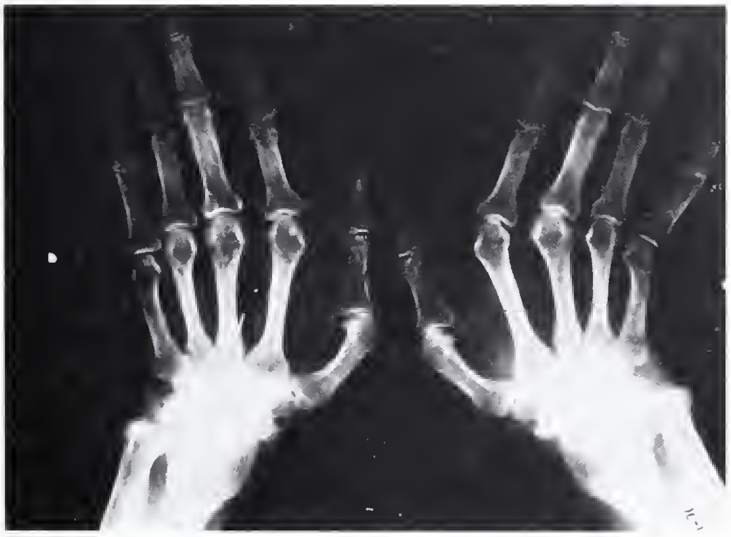

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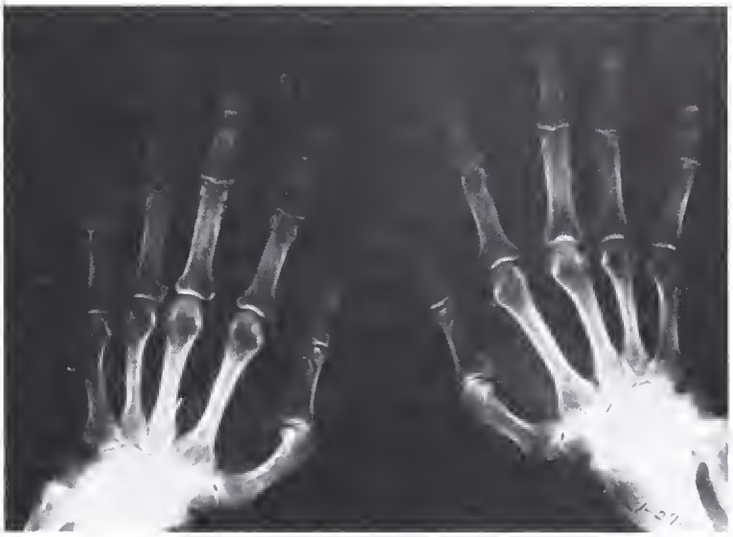

Figure 4

and hammer toes were present. The rest of the joint examination was normal. There was no clinical evidence of active synovitis. The back examination was normal. Rheumatoid factor was negative.

DISCUSSION

The prevalence of arthritic findings in the hands of the 85 patients is consistent with that expected from population studies. The occurrence of RA in 2 (2.4%) approximates the 1.0% for females and 0.5% for males found by Beasley in a survey of Alaska Eskimos (25). The 17 (19.5%) patients with DJD are less than that reported in the white population in the U.S. but corresponds to the 17% found by Blumberg in a survey of Alaska Eskimos (26). Hyperextension of the MCP's, noted in four patients without other evidence of arthritic disease, was also seen by Blumberg (26).

Jaccoud's arthritis occurs in the absence of an antecedent history of rheumatic fever (6) and as the sequelae of systemic lupus erythematosus (16). Patients with Jaccoud's arthritis and rheumatoid nodules (8) and with positive rheumatoid factor (5) have been described. Consequently, there still remains some question as to whether chronic, post-rheumatic fever arthritis is truly and entity, related primarily to the previous occurrence of severe rheumatic fever, or whether it is a variant of RA, perhaps with some modification of its presentation occurring as a result of the preceding rheumatic fever.

Bywaters, in 1950, (12) in a careful review of 27 autopsies on patients with RA, determined that there were three clinical relationships between RA and valvular heart disease.

1. RA with valvular deformity due to rheumatoid granuloma in the valves.
2. RA developing coincidentally in a patient who had also suffered an attack of acute rheumatic fever and developed rheumatic valvulitis.
3. An arthritic condition occurring in a person who has undergone several episodes of ARF with resultant chronic arthritis involving predominant-

ly the MCP joints. He felt there was good reason to feel that Jaccoud's arthritis could be clearly distinguished from a variant type of RA.

Thomas, in 1955, (2) reported retrospectively on 29 patients with valvular heart disease who had been followed in the rheumatism clinic at the Manchester Infirmary. Fourteen of the 29 had clear-cut RA. Eleven patients had experienced recurrent episodes of febrile polyarthritis. These patients demonstrated a much greater response to aspirin than did those with RA. Ten of the eleven showed joint changes with hypermobility of the MCP joints. There were no erosions, although several had effusions. Others (27-29) have further studied the cardiac findings in patients with chronic arthritic diseases.

Moro, in 1978, (13) reported on an evaluation of 400 patients with RHD. Seventeen patients had findings in the hands and feet fulfilling the criteria for the diagnosis of CPFA. All 17 of them identified a preceding episode of acute rheumatic fever and 10 were aware of more than one bout of ARF. Ten were female and the age range was from 23 to 64 years. The patients had two valves involved in 11 cases, the mitral valve only in one case. The patients had painless joint deformity with predominantly ulnar deviation of the MCP and lateral deviation of the metatarsalphalangeal joints. There were no signs of inflammation and all of the deformities were reducible.

Recently, the joint manifestations in a group of Mexican-American adults with rheumatic fever were described and termed "post-streptococcal arthritis" (30). None of the authors reporting Jaccoud's attempted to define a difference between the types of arthritis their patients presented with during their ARF, but a number of cases of Jaccoud's arthropathy (3, 4, 8, 9, 13, 14) as well as the present case had either their initial ARF or recurrent episodes of ARF as adults (after age 18).

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RISK MAN

Physician-Patient Communication

I was once asked by an attorney to review a medical record to determine whether malpractice had been committed by a gynecologist. I explained that I would review the record for the purpose of explaining the meaning of its contents to he and his client, but that I would not give any opinion concerning the presence or absence of malpractice. That was acceptable, and I spent some time carefully studying the chart with which I was provided. The client's wife, a woman in her late twenties, had recently given birth to her second child, after an unremarkable pregnancy and delivery. Following delivery of the placenta, slow but constant uterine bleeding was noted. Numerous efforts were made to stop the bleeding over the next several days, but all were unsuccessful. After learning that the couple did not plan to have additional children, her physician recommended a hysterectomy. The couple was attracted to that option, and agreed. A vaginal approach was suggested and agreed upon. Apparently, a discussion (not well documented) occurred in which the physician explained "the risks and possible complications" of the procedure. After appropriate preparation, the wife was taken to surgery.

The surgery was, according to the operative report, uncomplicated. A brief stay in the recovery room was completely uneventful. About two hours after returning to the floor, the patient began to complain of left flank pain. The pain, which was colicky in nature, worsened over the next several hours, and became constant. Urine output was decreased compared to the preoperative period. The pain continued into the night, and she was placed on analgesics.

The following day, the patient was again taken to surgery, and an exploratory laparotomy was performed. The left ureter was found to be caught in a suture. The entrapped portion of the ureter was resected, and the ureter was reanastomosed to the bladder. Postoperatively, two minor complications developed, but the patient went on to a full recovery. Renal function was preserved bilaterally.

After my review of the chart, I met with the attorney and the patient's husband. The wife did not attend the meeting. I asked the gentleman to tell me in his own words what happened. In a calm manner, he told me about the delivery, the subsequent bleeding, and the recommendation for a hysterectomy. The surgeon, he said, recommended a vaginal approach, "because then there wouldn't be any scar." The couple, according to the husband, was told that the procedure was straightforward, and that the wife would be able to go home "a couple of days after the surgery." He could not recall any discussion of risks such as infection, bleeding, or injury to neighboring organ systems. He could not recall discussion of alternatives to the proposed procedure.

In a straightforward manner, the husband continued to discuss the wife's course. He described the postoperative pain, and the return to surgery. At that point he became somewhat agitated. He stook up from his chair and began pacing. With increasing anger, he described the following: Neither he nor his wife were given an explanation as to the reason for the need for the second surgery. (In fact, he still did not know, even as he related the incident to me!) They assumed, however, that their physician would "take care of whatever it was." They were glad they had a good doctor. While his wife was in surgery, the husband remained in the waiting area. The doctor left the surgical area through a door to the waiting area. When the husband saw the physician, he stood and asked "how did it go?" The doctor replied, "Look, these things happen. That's just one of the risks of a hysterectomy." The doctor then rapidly strode from the room. The husband was confused, angry, and bitter. He had no further communication with the surgeon. The surgeon later explained to the wi that she had a "little problem" from th surgery, but that it was corrected, and sh should do well. Immediately after his wife discharge, the husband contacted an a torney.

As I observed the husband's demean and listened to his words, it became cle that the force propelling the husban toward a judicial attack upon the doct was not knowledge of medical wrongd ing, but rather anger and a perceived ci impropriety. He had no way to evalute th extent of the physician's knowledge, th expertness of his skills, or the reason bleness of his advice. Those facto played no significant role in placing th patient in hostile confrontation with th doctor. It was the surgeon's failure communicate concern and compassi for the wife's situation which destroye the physician-patient relationship.

Admittedly, the physician's behavior this case was, if true, more extreme th one might normally expect from a bus perhaps somewhat defensive doctor. graphically demonstrates, however, simple truism: anger is the common de nominator in most malpractice suits. doesn't matter if the incident did not occ exactly as the husband described something happened to trigger anger the part of the husband. What the doct actually said could be debated in a cou of law. But from a risk manageme standpoint, any debate in that particul forum is a debate which has gone muc too far. The practitioner who wishes manage risks must understand that p tient anger is the greatest single ri factor for litigation against the treati physician.

Now, for an exercise in risk manag ment, let us tinker a bit with the histo described above. Let's assume that th entire history remains the same—intrac able post-partum bleeding, a poor eff to obtain informed consent, an entrapp ureter, the second surgery, etc.—exce let us suppose the surgeon had respon ed differently to the husband. Imagine t following scenario: The husband stan

AGEMENT

l asks, "how did it go?" The doctor
ns, and replies, "Mr. Jones! It went well;
r wife is going to be just fine. I would
to discuss her situation with you in a
re private place, though. Do you have a
ute? Could we talk in my office?" The
walk to the doctor's office, or another
et area, and the physician offers the
band a cup of coffee. Then he contin-
: "The problem your wife was having
s the result of the first surgery. Some-
, in the process of performing the
terectomy, the tube connecting the
ney to the bladder was pinched by a
h. That blocked the flow of urine
ugh the tube, and the resulting pres-
e caused pain. We had to do a little
plumbing, but it looks like things are
ng to work out all right. If you like, I can
w you a sketch of what happened, or
w you on a diagram in one of my
ks, here. Naturally, I'll explain all of
to Mrs. Jones, but I wanted to explain
gs to you — and hopefully set your
d at ease — as soon as possible."

this scenario, the doctor has done
eral things. First, he has referred to the
band and wife by name, reaffirming
ecognition of them as unique individ-
. Second, he has demonstrated con-
in three ways: by taking time from a
schedule to talk to the husband, by
suming additional time taking the hus-
d to a location in which the husband is
e likely to feel at ease in asking
stions about his wife's condition, and
naking it clear that time was available
hose questions through the offer of a
of coffee.

his conduct, our imaginary doctor
nothing to prejudice his defense of
action which the family might bring
nst him. He did not admit liability. He
ot accept blame. He simply explained
t he found had happened.

am convinced that, in the case I
ewed, had the doctor communicated
h he may actually have felt, the family
ld not have consulted an attorney.
sider the following true case:

patient I know was hospitalized for
opedic surgery on a knee. The operation went entirely as expected, and no complications were encountered. The patient began several days of expected in-patient convalescene. Through an oversight, subcutaneous heparin was not administered. On the third postop day, the patient developed a pulmonary embolism. Anticoagulation was immediately begun, but the patient was in considerable distress for several days. The doctor was very apologetic, and thoroughly explained the situation to the patient, who did not sue. The patient explained to me that she held the doctor in high regard, and felt that he had made a mistake "like any of us might do." She did not wish to harm what she felt was a good relationship by suing the physician.

Clearly, no physician should ever count upon a similar response from a patient who has suffered from negligence. On the other hand, an appropriate communication of genuine concern will almost always be perceived by the patient, and the patient will usually be deeply appreciative. Almost certainly, the absence of a communicated concern for the patient will result in anger towards the physician, even if it is not expressed.

It is ironic that there is a too-infrequently utilized device on the market which can greatly reduce the incidence of malpractice suits. This device has been manufactured by a variety of suppliers, and is actually available in virtually all hospitals and doctors' offices. The product is called a "chair." When placed by a patient's bed or otherwise in close proximity to the patient, with no obstructions between the patient and docotor, and when regularly occupied by a physician who spends a few moments of each visit to communicate friendship and concern for the patient, its use will almost certainly result in a decreased risk of claims made against the physician.

Lee S. Glass, M.D., J.D. — has addressed numerous groups on risk management including the international College of Surgeons and the Department of Orthopedics, University of Washington School of Medicine. Glass practices law with Faulkner, Banfield, Doogan & Holmes in Anchorage and Seattle. He is also obtaining postgraduate medical training at the University of Washington.

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An Epidemic of Bullous Impetigo in a Newborn Nursery Due to Staphylococcus Aureus: Epidemiology and Control Measures

by W. Gary Hlady, M.D.[1]
John P. Middaugh, M.D.[2]

ABSTRACT

From January through August 1985, an epidemic of bullous impetigo caused by a penicillin-resistant strain of *Staphylococcus aureus*, phage type 3C, occurred in a newborn nursery in Anchorage. Of 479 infants at risk, 23 (4.8%) developed skin infections. There were no cases of septicemia, and no deaths occurred. Although the epidemic was temporarily interrupted by using hexachlorophene for bathing and handwashing, new cases occurred after hexachlorophene use was stopped. Epidemiologic investigation identified one nurse with active dermatitis on her hands, who had greater contact with case-patients than with controls at the onset of the outbreak ($p < .01$). Later during the course of the outbreak, contact with another staff member was associated with infection ($p < .05$), providing evidence of cross-contamination. Cases also occurred more frequently during times of high census in the nursery. The epidemic was finally terminated through vigorous enforcement of handwashing practices.

INTRODUCTION

Outbreaks of skin infections due to *Staphylococcus aureus* are a common problem in newborn nurseries and neonatal intensive-care units (1–3). Epidemics can occur either through the spread of infection by a staff member who is a carrier or by infant-to-infant spread from the hands of nursery personnel (4, 5). Identification of a carrier is based upon epidemiologic and bacteriologic information. This report describes a nosocomial outbreak of bullous impetigo caused by *S. aureus*.

BACKGROUND

The hospital, a 170-bed U.S. Public Health Service facility located in Anchorage and providing service primarily to the Native population, had an active obstetrical service. The mean number of live births per month in 1985 was 65 (range 31–83). All live infants who did not require intensive care were admitted to the newborn nursery. Infants who required intensive care were transferred to another unit.

"Rooming-in" of all well infants was encouraged and generally practiced, usually beginning within 12 hours after birth. Infants born by cesarean section began rooming-in on the third or fourth day. Infants were returned to the nursery at the mothers' convenience. The average length of stay for normal infants during the study period was 3 days. The average daily census was 8 (range 4–12), with an average of 4 (range 0–8) infants physically in the nursery.

Routine care of newborns before the outbreak consisted of an injection of vitamin K, 1% silver nitrate drops for prevention of neonatal opthalmia, and application of triple dye to the umbilical stump while the infant was in the delivery room. Infants were then transferred to a radiant warmer or isolette for the first 6 hours. The infants were removed at 2 hours for their first feeding and then were returned to the warmer. Infants were bathed daily with castile soap. Isopropyl alcohol (70%) was applied to the cord stump with each diaper change. Bathing and cord care were usually performed by the mother. Daily weighing and vital signs were taken by the staff. Nursery policy required handwashing with bactericidal soap only for the initial scrub. Castile soap was permitted for use between contacts with infants.

Four types of nursery staff were routinely exposed to newborn infants. The delivery-room nurse provided initial care and assessment immediately following delivery, as described above. She then transported the infant to the nursery where it was received by the nursery admitting nurse. Soon after their arrival in the nursery, infants were examined by the admitting physician. Prior to their release, all infants were examined by the discharge physician. Nursing staff worked rotating shifts and occasionally alternated their roles as delivery-room nurse or nursery admitting nurse.

Medical care of infants after discharge was usually

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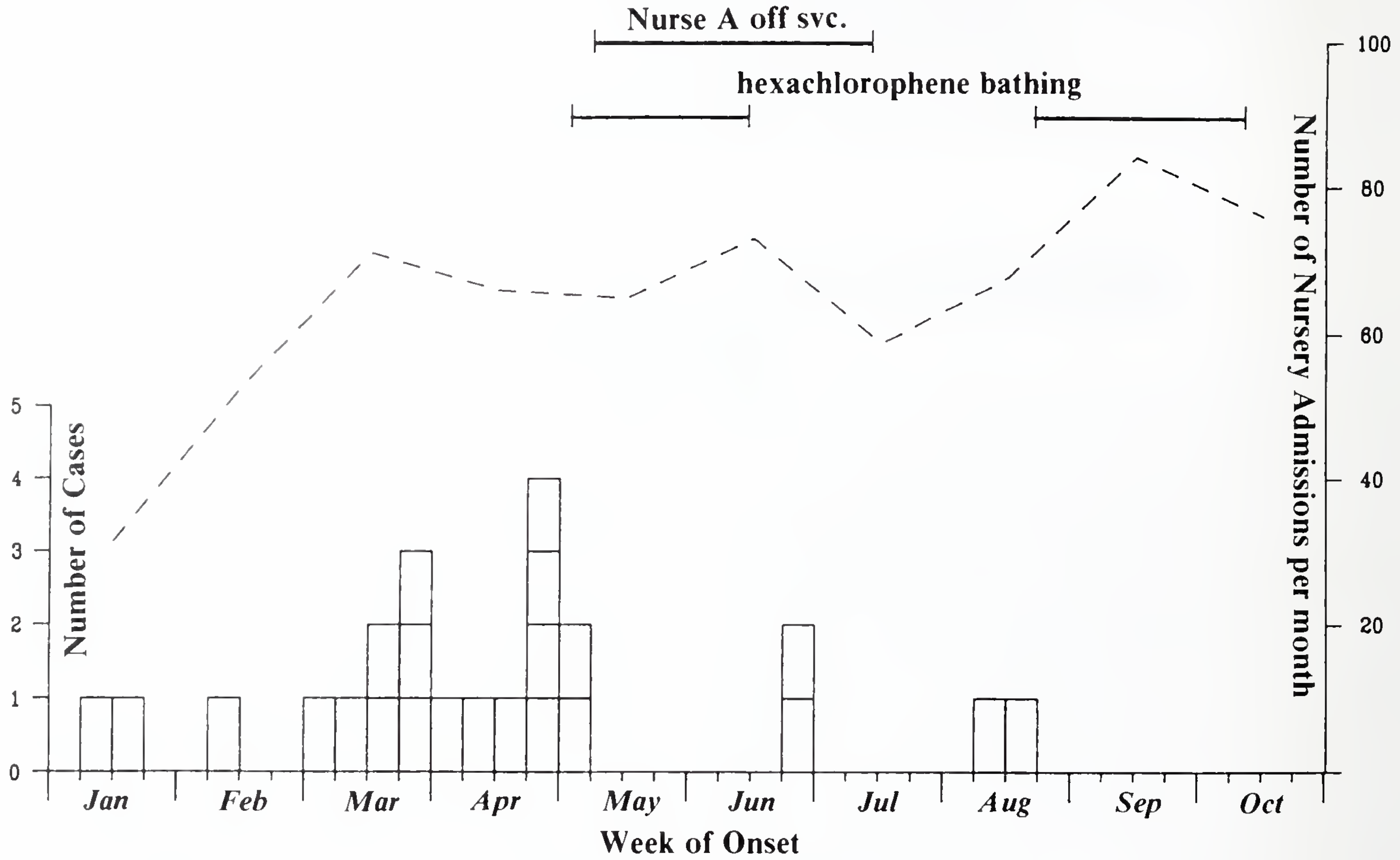
Figure 1

Weekly Incidence of Neonatal Staphylococcal Skin Infections,
Newborn Nursery, Alaska, January – October 1985



provided at Indian Health Service facilities.

DESCRIPTION OF THE OUTBREAK

From January 1 to August 31, 1985, 23 cases of neonatal staphylococcal skin infection were identified by active surveillance of laboratory results and physician reports (Figure 1). These 23 cases represented 4.8% of the discharges from the nursery (N = 479) during that period, a significant increase over the baseline rate of 0.5% observed over the previous 4 years ($p < 10^{-8}$, chi-square = 67.29).

Following the identification of seven cases during the third week in March, active surveillance and enforcement of handwashing policies were begun. During the next 5 weeks, ten additional cases were identified. On May 2, daily bathing of all infants with 3% hexachlorophene was begun and continued through June 13 (Figure 1). No new cases among infants born during that time were identified. Also during the first week in May, all nursery personnel were examined to clinically identify possible carriers and remove them from patient contact.

Four of the cases were identified within 9 weeks after hexachlorophene bathing was stopped. Daily hexachlorophene bathing of all nursery infants was reinstated on August 20 and was continued through October 10, during which time no new cases were reported (Figure 1).

CASE-CONTROL STUDY

Methods

A case was defined as any infant born at the hospital who had a pustule from which *S. aureus* was isolated during the first 28 days of life or who had a pustule that appeared staphylococcal but was not cultured, with onset within the first 28 days of life.

Hospital charts for all 23 cases and 44 controls were reviewed. The controls were selected by taking every ninth live delivery record from the delivery room log, excluding cases. The 44 controls represented 9.2% of all deliveries that occurred between January 1, 1985 and August 31, 1985. The significance of associations between exposure to hospital personnel and the development of disease was determined using either the Yates corrected chi-square or the Fisher exact test.

Results

The most common sites of infection were groin 35% (8/23), face 22% (5/23), and buttocks 17% (4/23). There were no cases of systemic staphylococcal infection. All but two of the case-patients were delivered vaginally; only 1 was preterm (37 weeks). Nine cases were in females and 12 were in males (4 were circumcised). The mean age at onset was 9 days with a range of 3 to 24 days.

The distribution by sex was nearly identical among cases (12 males, 11 females) and controls (24 males, 20 females). A larger proportion of control males (12/24) were circumcised. Cases and controls were both exposed to a median of six different hospital staff people.

The number and rate of cases per month appeared to increase with the number of nursery admissions prior to the institution of hexachlorophene bathing (Figure 1).

Inspection of all nursery personnel identified one, Nurse A, with dermatitis on her hands that began in December 1984. This person was one of three LPNs who had primary responsibility for the nursery. She was removed from the nursery from May 7 until her lesions cleared on July 12 (Figure 1). She received no antibiotics.

Exposures to the delivery room nurse, the nursery admitting nurse, the admitting physician, and the discharge physician were clearly documented for all cases and controls (Table 1). Cases were significantly more likely than controls to have been exposed to Nurse A ($p < .01$; O R = 4.96, 95% C I 1.68–14.65). Exposure to Nurse E was also statistically significant ($p < .05$; O R = 5.83, 95% C I 1.19–28.73), but only 22% of cases (5/23) were exposed to her. A significantly larger proportion of cases than controls was exposed to Nurse A when she was the delivery room nurse ($p < .01$; O R = 15.18, 95% C I 2.57 – 89.55) and the nursery admitting nurse ($p < .001$; O R = 8.22, 95% C I 2.73–24.81).

The difference between the proportions of cases and controls exposed to Doctor S as the discharge physician also reached statistical significance ($p < .05$; O R = 5.07, 95% C I 1.50–17.52). No other significant differences were found between exposure to cases and controls among 23 hospital personnel who attended infants in the four capacities for which clear documentation was uniformly available.

Because Nurse A, Nurse E, and Doctor S were exposures of interest, exposures to these three people were compared before and after the period, May 7 to July 12, when Nurse A was removed from the nursery. Cases were significantly more likely than controls to have been exposed to Nurse A only during the period when she had active dermatitis ($p < .01$, Yates corrected chi-square = 8.90; O R = 9.60, 95% C I 2.45–37.61). Exposure to Nurse E was also a significant risk factor prior to May 7 only ($p = .03$, Fisher exact test; O R = 10.00, 95% C I 1.43–70.06). Cases were significantly more likely than controls to have been exposed to Doctor S only after July 12 ($p = .02$, Fisher exact text, O R undefined). Two cases were diagnosed between May 7 and July 12; one had a documented exposure to Doctor S.

Two cases were not cultured for *S. aureus,* but the appearance of their lesions was typical. All bacterial isolates were resistant to penicillin and ampicillin; phage typing of staphylococci from five cases with onset before March 27 indicated that all were type 3–C. Only one of the isolates obtained from cases after March 27 was tested for phage type. It was obtained from a case with onset June 27 and it was not typable.

On October 9, 1985, cultures of nares, hands, axillae, perineum, rectum, and vagina were obtained from Nurse A. Staphylococci were isolated only from her hands. They were tested for phage type but were not typable. Cultures were not obtained from Nurse E nor Doctor S.

CONTROL MEASURES

On the basis of our findings, the following control measures were recommended on November 7, 1985:

1. Strict handwashing practices with a bactericidal soap (containing either 3% hexachlorophene, an iodophor, or 4% chlorhexidine) should be observed by all personnel exposed to infants in the nursery or on the obstetrical unit. Handwashing should include an initial 3-minute scrub to the elbows when reporting for duty, and a 15-second scrub (also with bactericidal soap) immediately before and after handling each infant, or after handling contaminated objects.
2. Personnel with active skin lesions should not be allowed to handle infants.
3. A record of contacts, including confirmation of handwashing, should be maintained for each infant at the cribside and made a part of the permanent patient record. Recording is intended to encourage compliance with handwashing guidelines and to facilitate epidemiologic investigation of future cases.
4. The number of different hospital personnel who come in contact with each infant should be minimized as much as possible.
5. Active surveillance for additional cases should be maintained and new cases reported immediately to the infection control nurse.
6. Routine bathing of infants with hexachlorophene is *not* recommended.

DISCUSSION

Epidemiologic findings suggest that the original source of the outbreak was Nurse A. The onset of the outbreak coincided with the onset of her dermatitis, and exposure to her was a significant risk factor for infection only during the time when her dermatitis was active. Failure to isolate the epidemic strain from Nurse A after her dermatitis had cleared is consistent with this finding.

There was some evidence that cross-contamination may have occurred between Nurse A and Nurse E early in the outbreak. And after Nurse A's dermatitis cleared,

Table 1

Proportions of 23 Cases and 44 Controls Exposed to Nursery Personnel Before the Onset of Disease by Person and Position, Staphylococcal Outbreak in a Newborn Nursery, Alaska, January–October 1985

	Delivery Rm Nurse		Nursery Admitting Nurse		Admitting Physician		Discharge Physician		Any★ Contact	
Personnel	**Case**	**Ctrl**	**Case**	**Ctrl**	**Case**	**Ctrl**	**Case**	**Ctrl**	**Case**	**Ctrl**
A	.26	.02**	.61	.06***					.74	.36**
B	.09	.07	.04	.07					.30	.18
C	.04	.02	.00	.02					.17	.20
D	.04	.09	.00	.11					.30	.32
E	.04	.05	.04	.02					.22	.05*
F	.09	.09	.04	.05					.17	.18
G	.09	.09	.00	.05					.22	.23
H	.13	.07	.04	.11					.30	.34
I	.04	.05	.00	.00					.04	.05
J	.04	.11	.04	.09					.13	.20
K	.04	.05	.00	.00					.04	.05
L	.04	.00	.00	.00					.04	.00
M	.04	.07	.09	.09					.22	.20
N	.00	.07	.04	.00					.13	.09
O	.00	.02	.04	.07					.65	.57
P	.00	.02	.00	.07					.70	.61
Q	.00	.02	.00	.00					.17	.14
R					.26	.20	.17	.39	.30	.52
S					.35	.20	.44	.14*	.52	.25
T					.17	.30	.17	.09	.22	.36
U					.13	.14	.06	.18	.17	.23
V					.04	.07	.06	.05	.09	.11
W					.04	.07	.11	.09	.13	.16

p-values were calculated by Fisher exact method (two-tailed) or Yates corrected chi-square.
*$p<.05$
**$p<.01$
***$p<.001$
★ Includes routine daily contact for weighing and vital signs.

exposure to yet another staff member was a significant risk factor. Also, cases were more likely to occur during periods of high census when strict handwashing practices may have been less likely to be practiced. In combination, these findings strongly suggest that cross-contamination occurred and played a major role in spreading infection.

Two cases occurred while Nurse A was off service, further evidence that cross-contamination occurred. Only one case was phage typed after March 27—the isolate was not typable. Although Nurse A also had a non-typable isolate cultured from her hands in October, epidemiologic investigation failed to demonstrate an association between cases and contact with Nurse A after her return. It is unlikely that Nurse A was involved in further transmission of staph in the nursery after her dermatitis healed and she returned to work on July 12.

Because subsequent isolates were not phage typed, we do not know if they were of the epidemic strain, 3-C. We do know, however, that infection during the last weeks of the epidemic was associated with contact with Doctor S and not Nurse A.

This outbreak illustrates the need to exclude personnel with active skin lesions from the nursery. It also stresses the importance of strict adherence to handwashing practices in limiting the spread of infection by cross-contamination.

The appropriate use of hexachlorophene in the control of staphylococcal disease in the nursery deserves special mention. Hexachlorophene bathing has been used to control and prevent staphylococcal illness among newborns for over 20 years, but there have also been reports of dermatologic, gastrointestinal, and neurologic disease attributed to the toxic effects of hexachlorophene (6–8). Detectable blood levels of hexachlorophene have been found in newborn infants after bathing and pathologic lesions have been found in the central nervous systems of rats and monkeys exposed to large amounts of hexachlorophene (9). In view of these findings, the Food and Drug Administration, American Academy of Pediatrics, and Centers for Disease Control (CDC) recommended, in 1972, that routine bathing of newborn infants with hexachlorophene-containing preparations should be discontinued (10).

Short-term, prophylactic daily bathing with 3% hexachlorophene may still be an appropriate and effective control measure during outbreaks of staphylococcal disease, but it should not supplant other basic control measures such as diligent handwashing, disinfection, and, where possible, cohorting (11).

During an outbreak, 3% hexachlorophene bathing should be restricted to infants with normal skin who weigh over 2500 g, with two daily applications (first 2 days) followed by careful rinsing. A 3% hexachlorophene or an iodophor preparation is safe and effective for routine handwashing of nursery personnel (10).

Although hexachlorophene bathing of infants might have contributed to control of this outbreak, it was not until strict handwashing with bactericidal soaps was rigorously enforced that the outbreak was finally terminated.

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J-6138 January 1986

Before prescribing, see complete prescribing information in SK&F CO. literature or *PDR*. The following is a brief summary.

* WARNING

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: **Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired.** If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: The bioavailability of the hydrochlorothiazide component of 'Dyazide' is about 50% of the bioavailability of the single entity. Theoretically, a patient transferred from the single entities of triamterene and hydrochlorothiazide may show an increase in blood pressure or fluid retention. Similarly, it is also possible that the lesser hydrochlorothiazide bioavailability could lead to increased serum potassium levels. However, extensive clinical experience with 'Dyazide' suggests that these conditions have not been commonly observed in clinical practice. Angiotensin-converting enzyme (ACE) inhibitors can elevate serum potassium; use with caution with 'Dyazide'. Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function. Thiazides may add to or potentiate the action of other antihypertensive drugs. Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

Supplied: 'Dyazide' is supplied as a red and white capsule, in bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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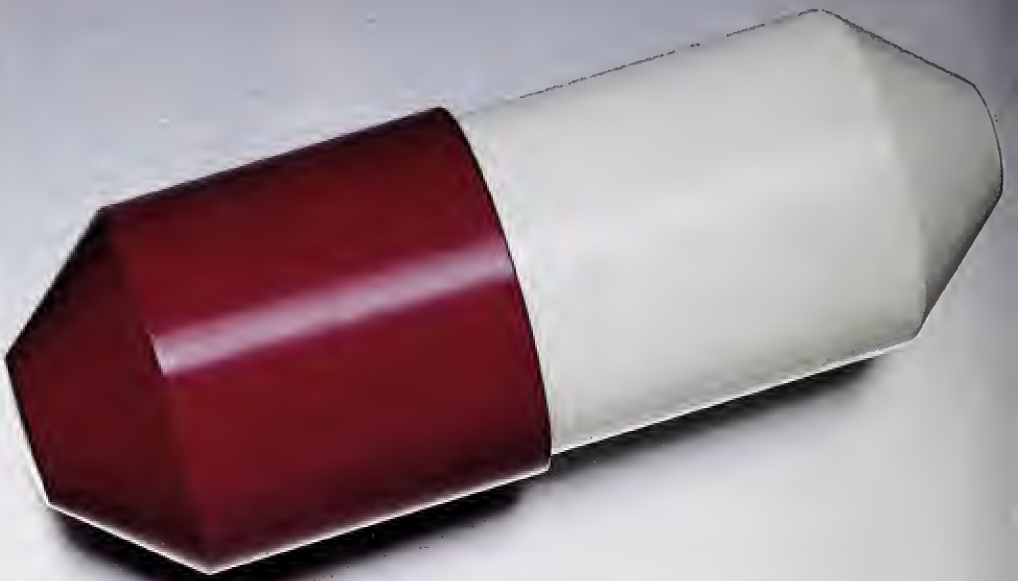

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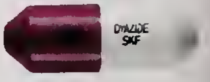

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CONTROL
ACID RAIN
with once-a-night
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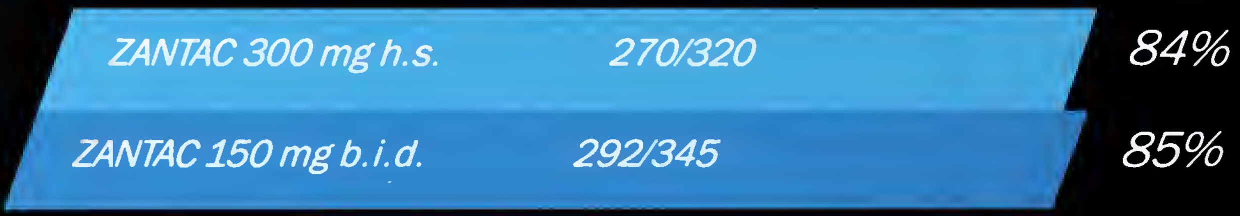
Only one tablet at bedtime

Controls nocturnal acid to relieve pain and heal duodenal ulcers

Heals active duodenal ulcers after 4 weeks in most patients[1]*



In well-controlled, double-blind, multicenter trials, ZANTAC 300 mg h.s. healed active duodenal ulcers in 84% of patients after 4 weeks. After 8 weeks, healing rates may be higher with ZANTAC 150 mg b.i.d. (92%) than with ZANTAC 300 mg h.s. (87%).

***Relieves pain and other symptoms as effectively as ZANTAC 150 mg b.i.d.**[1]*

Zantac® 300

ranitidine HCl/Glaxo 300 mg tablets

Once-daily dosing may enhance compliance in patients for whom dosing convenience is important

Side-effects profile comparable to ZANTAC 150 mg b.i.d.[1-3]

Headache—sometimes severe—has been reported. Rare effects on the CNS, cardiovascular, GI, hepatic, and integumental systems have been observed, as well as rare cases of hypersensitivity reactions. See ADVERSE REACTIONS section of Brief Summary of Product Information before prescribing.

No significant interference with the hepatic cytochrome P-450 enzyme system at recommended doses

ZANTAC 300 mg h.s. had no significant drug interactions with theophylline or warfarin. The bioavailability of certain medications whose absorption is dependent on a low gastric pH may be altered when ZANTAC or other medications which decrease gastric acidity are administered.

Glaxo/ROCHE

See next page for references and Brief Summary of Product Information.

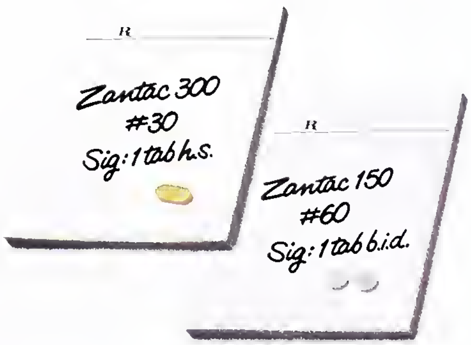
**It is not known exactly how much acid inhibition is needed to heal ulcers.*

IN ACTIVE DUODENAL ULCERS

Once-a-night h.s. therapy controls acid rain

Zantac®300
ranitidine HCl/Glaxo 300 mg tablets

Two effective regimens to treat active duodenal ulcers:



References: **1.** Data available on request, Glaxo Inc. **2.** Ireland A, Colin-Jones DG, Gear P, et al: Ranitidine 150 mg twice daily vs 300 mg nightly in treatment of duodenal ulcers. *Lancet* 1984,2:274-275. **3.** Colin-Jones DG, Ireland A, Gear P, et al. Reducing overnight secretion of acid to heal duodenal ulcers. *Am J Med* 1984; 77 (suppl 5B):116-122.

ZANTAC® 150 Tablets
(ranitidine hydrochloride)
ZANTAC® 300 Tablets
(ranitidine hydrochloride)

BRIEF SUMMARY OF
PRODUCT INFORMATION

See complete product information before prescribing. The following is a brief summary.

INDICATIONS AND USAGE: ZANTAC® is indicated in:
1. Short-term treatment of **active duodenal ulcer**. Most patients heal within four weeks.
2. **Maintenance therapy** for duodenal ulcer patients at reduced dosage after healing of acute ulcers.
3. The treatment of **pathological hypersecretory conditions** (eg, Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of **active, benign gastric ulcer**. Most patients heal within six weeks and the usefulness of further treatment has not been demonstrated.
5. Treatment of **gastroesophageal reflux disease (GERD)**. Symptomatic relief commonly occurs within one or two weeks after starting therapy. Therapy for longer than six weeks has not been studied.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain.

CONTRAINDICATIONS: ZANTAC® is contraindicated for patients known to have hypersensitivity to the drug.

PRECAUTIONS: General: 1. Symptomatic response to ZANTAC® therapy does not preclude the presence of gastric malignancy. 2. Since ZANTAC is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function. Caution should be observed in patients with hepatic dysfunction since ZANTAC is metabolized in the liver.

Laboratory Tests: False-positive tests for urine protein with Multistix® may occur during ZANTAC therapy, and therefore testing with sulfosalicylic acid is recommended.

Drug Interactions: Although ZANTAC has been reported to bind weakly to cytochrome P-450 in vitro, recommended doses of the drug do not inhibit the action of the cytochrome P-450-linked oxygenase enzymes in the liver. However, there have been isolated reports of drug interactions which suggest that ZANTAC may affect the bioavailability of certain drugs by some mechanism as yet unidentified (eg, a pH-dependent effect on absorption or a change in volume of distribution).

Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no indication of tumorigenic or carcinogenic effects in lifespan studies in mice and rats at doses up to 2,000 mg/kg/day.

Ranitidine was not mutagenic in standard bacterial tests (*Salmonella, E coli*) for mutagenicity at concentrations up to the maximum recommended for these assays.

In a dominant lethal assay, a single oral dose of 1,000 mg/kg to male rats was without effect on the outcome of two matings per week for the next nine weeks.

Pregnancy: ***Teratogenic Effects: Pregnancy Category B:*** Reproduction studies have been performed in rats and rabbits at doses up to 160 times the human dose and have revealed no evidence of impaired fertility or harm to the fetus due to ZANTAC. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: ZANTAC is secreted in human milk. Caution should be exercised when ZANTAC is administered to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Use in Elderly Patients: Ulcer healing rates in elderly patients (65 to 82 years of age) were no different from those in younger age groups. The incidence rates for adverse events and laboratory abnormalities were also not different from those seen in other age groups.

ADVERSE REACTIONS: The following have been reported as even in clinical trials or in the routine management of patients treat with oral ZANTAC®. The relationship to ZANTAC therapy has be unclear in many cases. Headache, sometimes severe, seems to l related to ZANTAC administration.

Central Nervous System: Rarely, malaise, dizziness, somnolenc insomnia, and vertigo. Rare cases of reversible mental confusio agitation, depression, and hallucinations have been reported, pr dominantly in severely ill elderly patients.

Cardiovascular: Rare reports of tachycardia, bradycardia, and pr mature ventricular beats.

Gastrointestinal: Constipation, diarrhea, nausea/vomiting, ar abdominal discomfort/pain.

Hepatic: In normal volunteers, SGPT values were increased to least twice the pretreatment levels in 6 of 12 subjects receivir 100 mg qid IV for seven days, and in 4 of 24 subjects receivir 50 mg qid IV for five days. With oral administration there ha been occasional reports of reversible hepatitis, hepatocellular hepatocanalicular or mixed, with or without jaundice.

Musculoskeletal: Rare reports of arthralgias.

Hematologic: Rare reports of reversible leukopenia, granuloc topenia, thrombocytopenia, and pancytopenia.

Endocrine: Controlled studies in animals and man have shown r stimulation of any pituitary hormone by ZANTAC and no antiandr genic activity, and cimetidine-induced gynecomastia and imp tence in hypersecretory patients have resolved when ZANTAC h been substituted. However, occasional cases of gynecomasti impotence, and loss of libido have been reported in male patien receiving ZANTAC, but the incidence did not differ from that in th general population.

Integumental: Rash, including rare cases suggestive of mild er thema multiforme, and, rarely, alopecia.

Other: Rare cases of hypersensitivity reactions (eg, bronchospasm fever, rash, eosinophilia) and small increases in serum creatinine

DOSAGE AND ADMINISTRATION: Active Duodenal Ulcer: The curren recommended adult oral dosage is 150 mg twice daily. An alte nate dosage of 300 mg once daily at bedtime can be used fo patients in whom dosing convenience is important. The advan tages of one treatment regimen compared to the other in a particu lar patient population have yet to be demonstrated.

Maintenance Therapy: The current recommended adult oral dosag is 150 mg at bedtime.

Pathological Hypersecretory Conditions (such as Zollinger-Elliso Syndrome): The current recommended adult oral dosage is 150 m twice a day. In some patients it may be necessary to administe ZANTAC® 150-mg doses more frequently. Doses should be adjuste to individual patient needs, and should continue as long as clini cally indicated. Doses up to 6 g/day have been employed i patients with severe disease.

Benign Gastric Ulcer: The current recommended adult oral dosag is 150 mg twice a day.

GERD: The current recommended adult oral dosage is 150 m twice a day.

See full prescribing information for dosage adjustment fo patients with impaired renal function.

HOW SUPPLIED: ZANTAC® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shape tablets embossed with "ZANTAC 300" on one side and "Glaxo" o the other. They are available in bottles of 30 (NDC 0173-0393-40 and unit dose packs of 100 tablets (NDC 0173-0393-47).

ZANTAC® 150 Tablets (ranitidine hydrochloride equivalent t 150 mg of ranitidine) are white tablets embossed with "ZANTAC 150" on one side and "Glaxo" on the other. They are available in bottles of 60 tablets (NDC 0173-0344-42) and unit dose packs of 100 tablets (NDC 0173-0344-47).

Store between 15° and 30°C (59° and 86°F) in a dry place. Protect from light. Replace cap securely after each opening.

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Glomerulonephritis Induced by Varicella

by Marvin E. Bergeson, M.D.[1]
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Lawrence C. Y. Agodoa, M.D.[2]

ABSTRACT

This report presents the occurrence of renal disease in a family following clinical and serologic varicella with a review of the literature associating varicella and renal disease since 1884. Renal disease among four siblings ranged from asymptomatic microscopic hematuria to hypertensive encephalopathy and acute renal failure. The acute renal failure progressed to chronic renal failure requiring chronic hemodialysis. Renal biopsy with light microscopy, immunofluorescence, and electron microscopy performed on three of the children showed varying degrees of proliferative glomerulonephritis. The acute nature of the renal disease in this family following varicella infection suggests post-varicella glomerulonephritis.

INTRODUCTION

Chickenpox is generally considered a benign disease of childhood. Complications, such as secondary bacterial infection, encephalitis, pneumonia, hypoglycemia, hepatitis, orchitis, arthritis, and the disseminated, progressive, hemorrhagic forms, occur primarily in the adult, and particularly in the immunocompromised host. (1, 2)

Although rare, renal disease following varicella infection including nephrotic syndrome, acute glomerulonephritis, and rapidly progressive glomerulonephritis has been described. In 1884 Henoch described four cases of varicella followed by "nephritic" urine with red cells, numerous cylindrical and epithelial cell casts, and albuminuria. These patients developed edema 8-14 days after the onset of varicella infection. (3) Two children with post-varicella renal disease as reported by Minkowitz (1968) showed glomerular hypercellularity on renal biopsy, and in one case, basement membrane thickening. Electron microscopy and immunofluorescence microscopy were not performed in these cases. Krebs and Burvant (1972) described an adult with nephrotic syndrome in association with varicella. Edema, weight gain, albuminuria, and proteinuria occurred two weeks after the onset of an acute illness characteristic of chickenpox. Renal biopsy revealed hypercellular glomeruli with proliferation of both endothelial and epithelial elements.(5) Recently Singhal, et al. (1977), described a 12-year old boy with rapidly progressive glomerulonephritis with five weeks of anuria commencing one week after the onset of a typical varicella infection. (7)

We described in this paper a family of four children afflicted with varicella infection, and renal involvement was documented in three of the four children.

MATERIALS AND METHOD

Three specimens of renal tissue were obtained by percutaneous needle biopsy utilizing the Travenol (R) disposable biopsy needle. Visualization was obtained with intravenous pyelography and fluoroscopy. In one case (MSR) retrograde pyelography was necessary for visualization. The first specimen was immediately frozen for immunofluorescence, and the other two were fixed in 6.25% glutaraldehyde solution buffered with sodium cacodylate. The frozen specimen was sectioned and stained with fluorescent antisera for IgG, C3, IgA and IgM. (8) One specimen fixed in glutaraldehyde was embedded in glycol methacrylate and stained with hematoxilin and eosin, and methenamine silver with Beibrich–Scarlet. (9) The other glutaraldehyde specimen was post–fixed in 1% buffered osmium tetraoxide and embedded in epoxy resin. Thin sections were then cut on the LKB III ultratome, stained for two hours with uranil acetate, then with lead citrate for five minutes. The sections were then examined with an AEI 801 electron microscope.

The skin biopsy was obtained from an uninvolved nonlight exposed area with a 4 mm punch. The specimen was fixed in 6.25% glutaraldehyde, buffered with sodium cacodylate, embedded in paraffin and examined by light microscopy. A second specimen was frozen, sectioned and stained with fluorescent antisera to human IgG, IgA, IgM, and complement C3 by the method of Lief, et al. (40)

Antiglomerular basement membrane antibody titers were determined by radio-immunoassay. (10) Serum immune complexes were detected and qualified by the

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[2] Departments of Pediatrics and Medicine, Madigan Army Medical Center, Tacoma, Washington

C1Q binding assay of Gabriel and Agnello. (11) Anti-streptococcal antibody tests were performed by the two minute hemagglutination slide tests (streptozyme by Wampole), and microtitration ASO-ADB system using commercially prepared antigen (Beckman). (12, 13) Acute and convalescent viral titers were performed by complement fixation technique utilizing commercially prepared antigen. Antinuclear antibody tests were accomplished by indirect fluorescent antibody method utilizing commercially available reagents.

Case #1

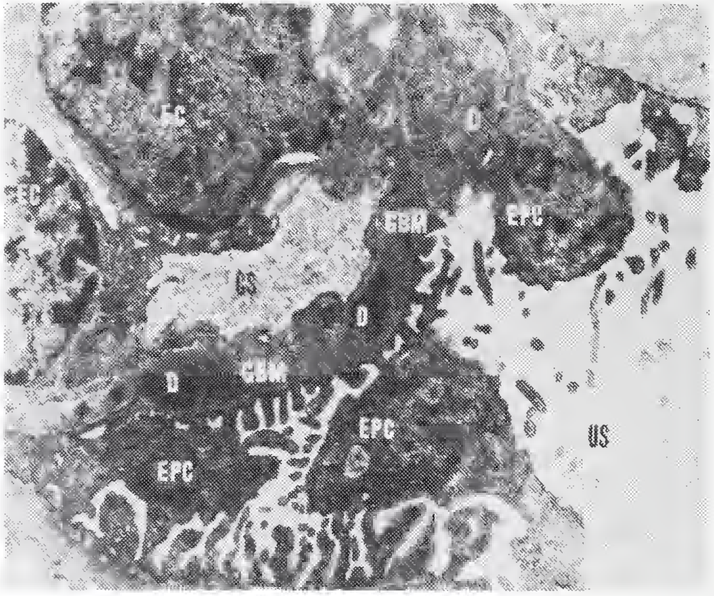
MSR is a 14-year old girl who presented in acute renal failure. Approximately two weeks prior to admission, she developed the symptoms and signs of chickenpox. The only medications she received were diphenhydramine hydrochloride (Benadryl) and trimethobenzamide hydrochloride (Tigan). She developed grand mal seizures two days prior to admission and on the day of admission.

Admission physical examination revealed a lethargic, but responsive patient with uremic odor, and blood pressure of 160/100. The skin showed the healing stages of chickenpox. Mild periorbital and pretibial edema were present. The deep tendon reflexes were symmetrically increased with ankle clonus.

Laboratory results are shown in Table 1. In addition, chest x-ray and the electrocardiogram were normal. CBC including normal platelet count was without evidence of intravascular hemolysis. Excretory urogram, renal scan and retrograde pyelography showed poorly functioning kidneys bilaterally, without evidence of obstruction. Urinalysis showed 4+ proteinuria, 20-25 WBC/hpf, TNTC RBC's, WBC, granular and RBC casts. The serum K was 7.4 and serum Ca 7.1 mg/dl. Serologic studies are listed in Table 2. The ANA was positive in a nucleolar pattern.

A percutaneous renal biopsy, performed following three weeks of anuria, showed by light microscopy 25 glomeruli present; none of which was obsolescent. All of the glomeruli showed diffuse proliferation, exudation, and epithelial cell proliferation with crescent formation. Approximately 60-75% of the tubules displayed atrophy and/or tubular damage. There was diffuse interstitial edema, fibrosis, and inflammatory cell infiltration with plasma cells, polymorphonuclear leukocytes, and occasional eosinophils. The vessels were normal. Immunofluorescent microscopy was negative for IgA, IgM, and IgG. There was diffuse granular deposition of C3 in the mesangium and on the peripheral capillary loops. Electron microscopy showed collapsed capillary loops, and electron dense deposits in the mesangium and within the lamina densa of the glomerular basement membrane. A skin biopsy was normal by light and immunofluorescent microscopy.

She was begun on peritoneal dialysis on admission and the hypertension was controlled on Aldomet, Inderal and Apresoline. She remains on dialysis.



LEGENDS

Figure 1 Electron micrograph of the renal biopsy of patient HR, showing intramembranous deposits. CS = Capillary space, D = Deposits, ED = Endothelial call. EpC = Epithelial cell, GBM = Glomerular basement membrane, US = Urinary space. Magnification x 6300.

Case #2

HR, the 11-year old, half-brother of MSR, was admitted with grand mal seizures two days following the admission of his sister. He developed the clinical signs and symptoms of chickenpox four weeks prior to admission which were treated with Tigan and Benadryl. He developed fever, nausea and vomiting one day prior to admission.

The admission physical examination revealed a temperature of 102 F., blood pressure 178/100, healing varicella skin lesions, and increased deep tendon reflexes with ankle clonus.

Laboratory results are listed in Tables 1 and 2. The chest x-ray and EKG were normal. The CBC including platelet count was normal without evidence of intravascular hemolysis. Urinalysis showed 3+ proteinuria, TNTC RBC's, 20-25 WBC/hpf, RBC, hyaline and granular casts. Immunoelectrophoresis showed normal levels of IgM and IgA with increased levels of IgG.

A percutaneous renal biopsy, performed one week after admission, showed by light microscopy eight glomeruli, six of which had diffuse intraglomerular cellular proliferation, moderate exudation, and a generalized diffuse increase in the mesangium. Two of the glomeruli showed parietal epithelial cell proliferation with crescent formation. There were mild diffuse interstitial edema and localized areas of inflammatory cell infiltration. Immunofluorescence demonstrated diffuse granular staining of all capillary loops and mesangium with C3, and mild staining with IgG. IgA and IgM were negative. Electron microscopy revealed granular electron dense deposits in the mesangium and in the glomerular capillary basement membrane (Figure 1).

The hypertensive crisis responded well to Hyperstat and hydralazine. At the two month follow-up evaluation he was normotensive on no medications and had normal renal function.

Case #3

JM, a nine-year old, half-brother of HR and MSR, was admitted for the workup of abnormal urinalysis. He had developed chickenpox two weeks prior to admission and received Tigan and Benadryl for the control of his symptoms. He was asymptomatic on admission.

The physical examination on admission was normal except for the resolving vesicular-papular exanthem of chickenpox.

Chest x-ray and EKG were normal. He had thrombocytopenia of 69,000/cu mm on admission which rapidly and spontaneously resolved. His CBC was without evidence of intravascular hemolysis. Urinalysis showed microscopic hematuria and RBC casts. Serum protein and immunoglobulin electrophoresis were normal. Laboratory results are shown in Tables 1 and 2.

A percutaneous renal biopsy, performed three weeks after the onset of the varicella, revealed by light microscopy 35 glomeruli present. All of the glomeruli showed diffuse intraglomerular cellular proliferation with mild exudation. No crescents were noted. The tubules contained RBC's, RBC casts, and occasional hyaline casts. Immunofluorescence demonstrated intense granular staining of the mesangium and capillary loops with C3. There was moderate granular staining of the mesangium with IgG. Mild IgA and IgM granular staining was present in the mesangium. Electron microscopy revealed electron dense deposits in the mesangium, and in the intramembranous areas of the glomerular capillary basement membrane. Mild mesangial sclerosis was present. The rest of the examination was normal.

His entire hospitalization was uneventful.

Case #4

MDR is the 12-year old, half-sibling of the first three patients. She was asymptomatic on admission. She had clinical varicella two weeks prior to admission. Trimethobenzomide hydrochloride and diphenhydramine hydrochloride were used in treating her abdominal cramps and pruritus.

Physical examination was normal except for the healing skin lesions of chickenpox.

Laboratory studies are shown in Table 1 and 2. CBC was without evidence of intravascular hemolysis. Serum protein and immunoglobulin electrophoresis were normal. The skin culture was positive for beta-hemolytic streptococcus (Group A).

At one- and two- month follow-up examinations, she was asymptomatic with a normal urinalysis. A renal biopsy was not performed in this patient.

COMMENTS

In the past decade much has been written about the induction of renal diseases by viruses (4-7, 14, 17). Approximately eighteen viruses, including Coxsackie, Echo, and Mumps, have been implicated in the literature in the induction of glomerulonephritis. (15) Varicella was first described to cause nephritis by Henoch in 1884. (3) In their review of the literature through 1928, Denney et al., reported 52 cases of acute nephritis associated with varicella. (16)

The incidence of varicella induced renal disease is quite low. A prospective study examining patients' urines two weeks after clinical varicella was done by Cassel in 1883. He found one case of "nephritis" in 200 cases of varicella. (18) The largest study of complications associated with varicella reported three cases of nephritis out of 2,534 patients with post-varicella complications. (2) A smaller series found a single case of nephritis among 775 patients. (1) The largest outbreak of virulent varicella nephritis occurred in the French Cameroon in 1936 where 370 patients died of nephritis out of 1919 patients hospitalized with varicella. (19) Complete data including serology and renal biopsy studies are, however, lacking in all of the above studies.

The four children reported here developed a spectrum of renal disease 10-14 days after the onset of clinical varicella. MSR developed anuria, hypertension, seizures, and stupor. HR manifested gross hematuria, azotemia, severe hypertension, and seizures. The other two children were asymptomatic although JM had microscopic hematuria. The subsequent clinical course for each child was distinctive in that MSR progressed to chronic renal failure, HR recovered full renal function, and JM's renal disease did not progress beyond transient microscopic hematuria.

Viral studies (Table 2) showed elevated titers to varicella in all of the four children. A significant drop from acute to convalescent titers is noted in all but HR who was already in a convalescent state at the time of the evaluation. Titers to other viruses including Coxsackie,

Table 1. Laboratory Results

	Case 1 MSR	Case 2 HR	Case 3 JM	Case 4 MDR
Hemoglobin (mg/dl)	6.3	10.1	13.8	13.8
White Cell Count (x 10^3/mm^3)	13.3	17.5	7.0	8.0
Erythrocyte, sedimentation rate (mm/hr)	76	116	40	24
C3* (mg/dl)	33	46	80	96
C4** (mg/dl)	55	31	26	36
CH-50*** (units)	63	23	77	74
Antistreptolysin 0 (acute) titer	680	340	960	240
Antistreptolysin 0 (convalescent - 4 week) titer	240	240	480	170
AntiDNAse B (acute) titer	960	680	480	680
AntiDNAse B (convalescent - 4 week) titer	680	480	480	480
Throat culture	Negative	Negative	B. Hemolytic Strep	Negative
Skin culture	Negative	Negative	Negative	B. Hemolytic Strep
Antinuclear Antibody	1:1280	1:80	Negative	Negative
Rheumatoid factor	Negative	Negative	Negative	Negative
DNA binding %	8	8	3	0
Circulating Immune complex (mcg/ml)****	850	>1000	Negative	Negative
Antiglomerular basement membrane antibody titer	Negative	Negative	Negative	Negative
HAA	Negative	Negative	Negative	Negative
VDRL	Negative	Negative	Negative	Negative
BUN (mg/dl)	255	68	11	14
Serum creatinine (mg/dl)	29	1.8	0.7	0.5
Creatinine clearance (ml/min/m^2)	<5	17	79	74
Total protein (mg/dl)	8.1	7.8	8.0	7.4
Albumin (mg/dl)	2.8	4.0	4.7	4.0

*Normal 55-120 mg/dl
**Normal 20-40 mg/dl
***Normal 60-161 units
****Normal 20 mcg/ml

Echo, and Mumps were negative.

Serological studies showed positive ANA titers in MSR and HR. This led to the consideration of Systemic Lupus Erthematosis (SLE) as a possible etiology in the pathogenesis of the renal disease. Both patients, however, failed to demonstrate any of the typical criteria for the diagnosis of SLE. (41) DNA-binding and LE preps were negative. Skin biopsy of MSR did not show the characteristic positive band test (40). It is suspected that the positive ANA may represent a general immunological stimulation by herpes virus. (27) Animal studies indicate that an elevation in ANA can be induced by viruses. (28-31) In humans positive ANA has been reported with acute hepatitis (31-34) and infectious mononucleosis (27, 31, 32, 35-37). Chronic infections such as malaria and leprosy also may induce a positive ANA. (37-39)

Infectious and autoimmune disorders such as SLE are known to activate the complement cascade. In two of the patients (MSR and HR), total hemolytic complement and C3 were low; but, C4 was normal (Table 1). This suggests that their renal disease is probably associated with the alternate complement pathway. Previously described cases of varicella with renal disease have reported either depressed or normal complement levels but without the description of the specific pathway involved. (6,7).

Two of the four patients (MSR and HR) also demonstrated markedly elevated circulating immune complexes, a phenomenon which is nonspecific for any pattern of renal disease. Elevated circulating immune complexes have been reported in patients with various renal and systemic diseases including membranoproliferative glomerulonephritis (20,21), post-streptococcal glomerulonephritis (20), SLE (22,23), dengue (24), dermatitis herpetiformis (25), juvenile rheumatoid arthritis (20), and others.

All the children who manifested clinical renal disease had kidney biopsies performed. On light and electron microscopy, all glomeruli showed hypercellularity and exudation. Biopsies of MSR and HR showed parietal epithelial cell proliferation with crescent formation. The nearly total crescentic involvement of MSR's glomeruli is particularly significant and is similar to the varicella induced renal disease described by Singhal, et al. in 1977. (7) Although crescent formation in glomeruli may be seen in various renal diseases such as membrano-proliferative glomerulonephritis, post-streptococcal glomerulonephritis, Henoch-Schonlein purpura, SLE, polyarteritis nodosa, and the hemolytic uremic syndrome (26), crescentic glomerulonephritis is most commonly seen in Goodpasture's syndrome and rapidly progressive glomerulonephritis. The latter two diseases are usually associated with elevated circulating anti-glomerular basement membrane antibodies and linear immunofluorescent staining. None of the patients had detectable circulating antiglomerular basement membrane antibody levels, and the biopsy specimens did not show linear immunofluorescent staining.

Of particular interest in the determination of the pathogenesis of the renal disease in these children is the associated positive ASO and ADB titers (Table 1). Skin cultures were positive for beta-hemolytic streptococcus only in MDR who had normal ASO titers and elevated ADB, but showed no clinical evidence of renal involvement. JM showed positive throat cultures for Group A, beta-hemolytic streptococcus, and he had very mild renal disease both clinically and by pathology. Although not documented by positive cultures, it would not be unusual for superinfection of the varicella skin lesions with streptococcus. M-Protein typing of the streptococci was not done in the two positive cases to determine whether or not the nephritogenic strains were encountered. The renal biopsy findings in these patients did not reveal the typical lumpy bumpy immune complex deposits on the glomerular capillary basement membrane characteristic of acute post-streptococcal glomerulonephritis. Instead, predominantly mesangial and intramembranous deposits are found in the biopsy specimens of all of the three patients.

It is our contention from the clinical and laboratory data presented in this paper that the renal disease in these children was induced by varicella. The acute onset of their syndromes, the associated clinical and serological manifestations of varicella infection, and the pattern of immunological and histopathological changes, suggest this agent as the cause of their glomerulonephritis. The full clinical spectrum and precise incidence of post-varicella glomerulonephritis can only be determined by well-planned prospective studies.

Table 2. Serologic Studies

Viral Titres

	MSR		HR		JM		MDR	
	A	C	A	C	A	C	A	C
	NEG		NEG		NEG		NEG	
Varicella	160	80	40	40	160	40	160	40
Herpes Simplex	80	80	5	<5	<5	<5	<5	<5
CMV	10	5	5	5	<5	<5	5	5
Echo type 4	5	<5	10	<5	<5	<5	<5	<5
Coxsackie	<5	<5	10	<5	<5	<5	<5	<5
Rubella	16	8	<8	<8	8	8	64	64
Rubeola	5	10	10	<5	10	5	20	10
Mycoplasma			5	<5			<5	<5
Toxoplasma			<40	<40	<40	<40	<40	<40
Adenovirus	5	<5	5	<5	<5	5	5	<5
Influenza A			5	<5			<5	<5
Influenza B			5	<5			<5	<5
Arbo California	5	<5	5	<5	<5	<5	<5	<5
Arbo St. Louis	10	<5	5	<5	<5	<5	<5	<5
Arbo Western	5	<5	5	<5	<5	<5	<5	<5
Arbo Eastern	5	<5	5	<5	<5	<5	<5	<5
Febrile Agglutinins	NEG		NEG		NEG		NEG	

A = Acute
C = Convalescent (4 weeks)

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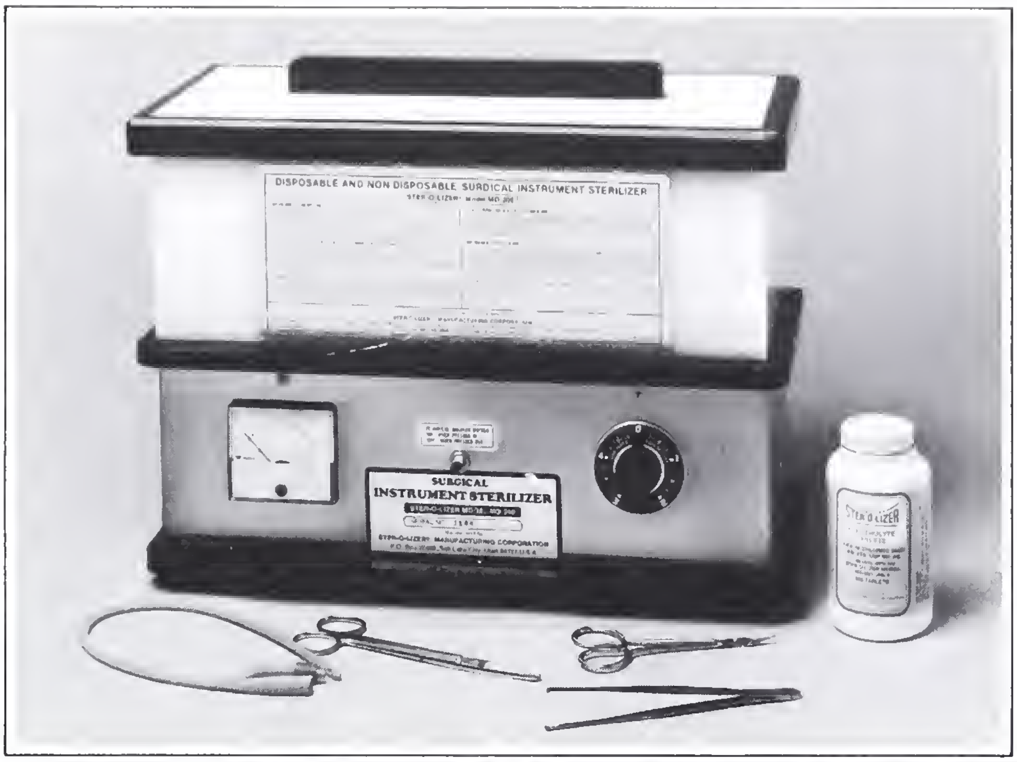
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Hepatitis B "e" Antigen in Individuals Infected with the Aids Virus

by Elizabeth A. Tower, M.D. and John P. Middaugh, M.D.[1]

The Alaska State Division of Public Health in May 1985 established an Alternate Site, Human T-cell Lymphotropic Virus Type III/Lymphadenopathy-Associated Virus (HTLV-III/LAV) Screening Program. We have observed a relationship between the presence of HTLV-III/LAV antibody and hepatitis B "e" antigen (HBeAg) positivity in homosexual and bisexual men who show evidence of coexistent infection.

Between June 1985 and June 1986, 937 sera submitted for HTLV-III/LAV antibody screening at alternate test sites in Alaska were screened for evidence of hepatitis B virus (HBV) infection and HTLV-III/LAV antibody by enzyme-linked immunosorbent assay. Male homosexual activity was identified as a risk factor in 333 men who were tested for reasons other than routine blood donation; 89 (27%) were HTLV-III/LAV antibody positive, 11 of whom also were positive for hepatitis B surface antigen (HBsAg). Of the 11 positive for both HBsAg and HTLV-III/LAV antibody, 10 were HBeAg positive; none had hepatitis B "e" antibody (anti-HBe). Of the 244 men who were negative for HTLV-III/LAV antibody, five were HBsAg positive; three of the five had developed anti-HBe (Table 1).

Homosexual or bisexual men showed a high prevalence of past HBV infection. Of 89 who were positive for HTLV-III/LAV antibody, 70 (79%) had HBV markers compared to 138 (56%) of 244 who were HTLV-III antibody negative (Table 2).

Of 604 persons not identified as having had male homosexual or bisexual activity as a risk factor, none of the 16 who were positive for HTLV-III/LAV antibody were HBsAg positive. HBsAg was present in 25 (4.3%) of the 588 who were negative for HTLV-III/LAV antibody; four of whom were positive for HBeAg, probably associated with recent HBV infection (Table 3).

Markers of past HBV infection were less prevalent in this group than in homosexual or bisexual males. Of 16 who were positive for HTLV-III/LAV antibody, six (37.5%) had HBV markers compared to 133 (22.6%) of 588 who were HTLV-III/LAV antibody negative (Table 4).

The State of Alaska began in 1983 a statewide, hepatitis B screening and immunization program. We identified an additional 26 homosexual males who were positive for HBsAg but have not yet been screened for HTLV-III/LAV antibody.

HBeAg is present in 19 (73%) of these men; in 13, HBeAg has persisted on repeat testing during the three-year period. None of those who were HBeAg positive have developed anti-HBe.

No information is available at this time regarding the temporal relationship between hepatitis B infection and HTLV-III/LAV infection in any of these people. In view of the impairment in T-cell immunity in persons with HTLV-III/LAV infection, it is not surprising to observe a high prevalence and persistence of HBeAg, believed to correlate with acute hepatitis B viral replication, in those positive for HTLV-III/LAV antibody.

Persons infected with both HTLV-III/LAV and HBV may be highly infectious for hepatitis B for a prolonged period of time. Efforts should be increased to vaccinate susceptible homosexual and bisexual men and their contacts against hepatitis B. Health care workers, particularly those who care for these individuals, should receive hepatitis B vaccine.

Table 1. HBsAg and HTLV-III antibody in Homo- or Bi-sexual Males in Alaska, 1985-1986.

HTLV-III Antibody

		Positive	Negative	Total
HBsAG	positive	11*	5☆	16
	negative	78	239	317
Total		89	244	333

Chi-square = 15.16
$p = 9.87 \times 10^{-5}$

* Of 11 positive for HTLV-III and HBsAg, 10 were positive for HBeAg.

☆ Of 5 negative for HTLV-III and positive for HBsAg, 1 waspositive for HBeAg.

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[1] Epidemiology Office, Division of Public Health, 3601 C St., Suite 540, P. O. Box 240249, Anchorage, AK 99524-0249

Table 2. Hepatitis B virus (HBV) markers and HTLV-III antibody in Homo- or Bi-sexual males in Alaska, 1985-86.

		HTLV-III Antibody		
		Positive	Negative	Total
HBV	positive	70	138	208
	negative	19	106	125
Total		89	244	333

Chi-square = 13.58
$p = 2.286 \times 10^{-4}$

Table 3. HBsAg and HTLV-III antibody in other than Homo- or Bi-sexual males.

		HTLV-III Antibody		
		Positive	Negative	Total
HBsAG	positive	0	35	25
	negative	16	563	579
Total		16	588	604

Chi-square = 1.11
p = 0.29

Table 4. Hepatitis B virus (HBV) markers and HTLV-III antibody in other than Homo- or Bi-sexual males.

		HTLV-III Antibody		
		Positive	Negative	Total
HBsAG	positive	6	133	139
	negative	10	455	465
Total		16	588	604

Chi-square = 1.95
p = 0.163

Announcement

The Annual Meeting of the United States-Canadian Division of the International Academy of Pathology will be held at the Palmer House in Chicago, Illinois starting with "Companion Meeting" symposia on Sunday, March 8 and continuing through Friday evening, March 13, 1987.

Scientific Papers, Poster Sessions, thirteen Specialty Conferences, and 44 Short Courses are scheduled. Two Special Courses will be offered on "Diagnostic Cellular and Molecular Pathology" with Dr. Benjamin Trump as Course Director and "Advances in the Application of Immunoctyochemistry to Diagnostic Surgical Pathology" with Dr. Cecilia Fenoglio-Preiser as Course Director. The Long Course will be on "Pathobiology and Recognition of Malignant Melanoma" with Drs. Martin Mihm and George Murphy as Course Directors.

The Maude Abbott Lecture will be delivered by Dr. Emmanuel Farber on Tuesday, March 10.

Timely Topics in Pathology will feature a session on "Telepathology - Delivery of Pathology Diagnostic Services in the Future" presented by Dr. Ronald S. Weinstein.

A special lecture entitled "The Changing Faces of Pathology and Medicine" will be given by Dr. Peter Ward.

Further information about the meeting and courses may be obtained from Dr. Nathan Kaufman, Secretary-Treasurer, United States—Canadian Division of the International Academy of Pathology, Building C, Suite B, 3515 Wheeler Road, Augusta, GA 30909. Telephone (404) 733-7550.

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History of Medicine in Alaska

ROBERT D. WHALEY, M.D.

Bob was born in California which has nothing to do with the fact that he can describe himself as a chemist, physicist, geologist, meteorologist and pilot.

Having received a degree from the University of California, Berkeley in seismology and physics in 1942, he worked with the weather bureau during World War II, after which he became a graduate student in psychology and began pre-med intending to become a psychiatrist.

He met Helen Stoddard. They contrived to get into the same medical school, University of California, San Francisco. Helen and Bob were married and were graduated in 1950. While they both entered internal medicine, Helen later changed to pediatrics to avoid conflicts of competition.

Having been drafted into the Air Force in 1953, Helen and he were separated for a year. She did pediatrics at Denver General Hospital while he was stationed in Anchorage. Before he finished his tour of duty, Helen came to Anchorage where she joined the Anchorage Medical and Surgical Clinic. At this time, the clinic comprised Howard Romig, Asa Martin, Jack Sedwick and Bob Wilkins. There was a polio epidemic in Anchorage in 1954. These were the days of the iron lung.

Following his stint in the Air Force, Bob and Helen went off to Boston where he had one year's training in pulmonary disease at Peter Bent Brigham Hospital. Helen did pediatrics, training in neurology with Dr. Lennon, a renowned physician, especially in the field of EEG. This was to spur her interest in brain damaged children.

Having returned to Anchorage, Helen went into practice with John Tower then having office and residence on 2nd Avenue in one of the old railroad houses. Bob spent two years in the Public Health Service with the Arctic Health Research Institute. He flew himself around the state working in cardiology. One December he was forced down on the Kuskokwim and had to walk eight miles into Red Devil. However, his favorite story has to do with attempting to tie down his plane at Shishmaref. He found some lovely clear ice and began to cut a fine U shaped hole much to the amusement of the Eskimos. Brushing the ice chips from his eyebrows, he learned that this was the cesspool.

Bob joined the Doctor's Clinic with William Ivey, Vern Cates, Winthrop Fish, Lester Margetts* and Merritt Starr*. Bob Whaley says now that "there were all too few doctors then. We were putting out fires. We made $8-12,000 a year."

From 1965-67, the Whaleys spent two years in California. Helen had a Neurology Fellowship at Stanford. Since this was to be for only a year, Bob took a sabbatical. However, when the Fellowship stretched another year, Bob Whaley became convinced that "I always want to work. I get depressed not working. My plan was to work indefinitely but not feel driven." He returned to independent practice in internal medicine.

Helen was diagnosed with breast cancer. She was to die in 1971. Before her death, she worked especially with brain damaged children and very much wanted a school started for them. Named for her, the Helen S. Whaley Center was opened in 1972. She probably would never have allowed this while alive. She was unusually intelligent, perceptive and modest but demanding of herself and others.

Bob recalls being radioed from Flat Horn Lake in 1960. An eight year old child was convulsing with a high fever. Bob took Helen in his airplane. It was dark in September but in spite of the blackness, he landed on the lake. The boy had a 105° temperature and a strep throat, quite like scarlet fever. It was one of the Reddington boys later of dog racing fame.

In the early sixties, Herb Shaindlin, always a promotor of controversy, had a radio program. For weeks he talked about there being no doctors in the hospitals at night. All doctors saw patients as needed but none stayed at the hospitals. A step forward was the advent of the "Little Red Wagon" which took emergencies to the hospitals at all hours.

There was a community wide effort of the medical profession and many others to get the whole population of Anchorage immunized with Salk polio vaccine. This was accomplished partly because a city of less than 50,000 can be reached; mainly because Bob organized superbly. He was honored as Physician of the Year in 1963. He was to receive this award again for his work on the malpractice problem of 1976.

About the time of the earthquake, Bob and George Hale did a cardiac resuscitation, the first in Alaska. They defibrillated a man who, in the process of a routine examination, had his heart stop. The patient survived six or eight years. About the same time, Bob did the first atrial fibrillation conversion.

***deceased**

During the earthquake of 1964, Bob recalls that the Community Hospital[1] had broken gas lines and no power. Patients were taken to Providence Hospital which only a few years before had been rebuilt in the "boonies". The Whaleys lost their house which had been built on Turnagain Bluff. This author remembers being in that house and having Bob, with his usual exactness, point out that the Cook Inlet flowed by at about sixty-five miles an hour (he did not use knots) and was severely undercutting the bluff.

Some years later, a DC-8 carrying military personnel and their dependents to Korea went off the runway at the Anchorage International Airport when the brakes froze. There were eighty to ninety passengers and crew seriously injured with burns and fractures. Numerous x-rays were needed and much warming up since it was November. The civilian medical community was called upon to assist with this crisis.

Bob Whaley has been president of both the Alaska Heart Association and the Alaska State Medical Association.

Bob married Jean Persons, M.D. in 1973. She brought, along with shared values and interests, three daughters. Bob has enjoyed becoming a stepfather. It is a satisfaction to him that one of them took his name. Jean has her own story to be told later.

Bob had intended to keep on practicing. However, he found the red tape, government regulations and the pressures of medical liability too much so he retired on January 1, 1986.

Since retirement, he has been working for the Social Security Disability Determination program. He continues to fly at which he is experienced and skillful. He likes motorcycles, and Jean and he make tours of far away places on them. Switzerland is a special favorite for this activity.

Bob has stong comments to make on the current socio-economic changes in medicine. "I think the worst thing in medicine has been the gradual development of third party medicine. In the 50's and 60's, it was called health insurance, but it was really prepaid medical care. During the next fifteen years, doctors were in the luxurious position of not having to worry about medical costs since the patient did not pay. The total cost sky-rocketed from 4-5% to 12% of the Gross National Product. The result is that even if a person is financially responsible, he can not afford medical costs. It's even more expensive to die. Doctors have lost control, and agencies and government are dictating medical practices."

He finds it less interesting to practice. "There is too much technology. A patient comes in with a syndrome, and we plug him in to algorithim from A to Z. It's cook book stuff. We used to have to go on intuition and hunches. You had to know medicine; had to notice the fine subtle things; then go to treatment from what you had surmised. It was like doing a puzzle. It was fun but never trivial."

The interviewing of Bob Whaley and Jean Persons was done by Ruth Nighswander.

Gwynneth Gminder Wilson
ASMA Auxiliary

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[1] Started by the Presbyterian Church, turned into the Community Hospital, bought by the Teamsters, it was located at 825 L Street until moved to its present location now Humana Hospital.

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March 25, 26, 27

For the first time, more than 30 of the Pacific Northwest's leading specialists in the field of geriatric care join in a symposium aimed at making nurses and other health-care professionals more expert in caring for the elderly.

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Editorial . . .

On AMA's Interest in Public Health

By William Foege, MD, in *The Nation's Health:*

It would have been easy to believe I was attending an American Public Health Association meeting. As I listened to the debate at the American Medical Association's House of Delegates meeting in June, two observations repeatedly intruded. First, much of the discussion concerned public health, and second, many of the resolutions and reports, relating to public health issues, resulted from initiatives taken by medical students and/or residents.

Concerning the first, public health concerns consumed more time and thought than any other item. There was debate on AIDS and ways of decreasing the rate of transmission. Multiple resolutions on tobacco provided the AMA an opportunity to strengthen the increasingly strong stand it is taking against tobacco advertising and tobacco use. Special note was made of the dangers of "designer" brands which, by implication, give smokers instant sophistication. The AMA is also beginning to look at alcohol advertising and the resulting adverse health effects. The delegates took a particularly encouraging approach to automobile safety, advocating the use of seat belts and airbags as the safest approach. Reports on child abuse, elder abuse, acquired immune deficiency syndrome, and school discrimination all emphasized the concern for prevention and public health.

The second observation, the role of students and residents, holds great promise for the continuing involvement of the AMA in public health issues. It reflects the social activism reported from many medical schools and builds on the significant, even spectacular, increases in AMA prevention activities even beyond the House of Delegates meetings. . . .

What are the implications of this trend? Without question, the health of people is enhanced. Public health, in the past, rarely received the support required and greatly benefits from the power and prestige inherent in the AMA. But medicine also needs this association. Concern for prevention and public health makes medicine more complete. It demonstrates an interest in people, not just their illnesses. It involves practitioners in the entire spectrum from health promotion through disease prevention to disease treatment, much as dentists have for years been the chief advocates of fluoridation and caries prevention, rather than just caries treatment.

This is just one aspect of a trend that appears to be accelerating, namely, public health is not only becoming a significant part of the social agenda but it is becoming everybody's business, not just health professionals, but educators, PTAs, journalists, unions, employers, insurance companies, advertisers, the Supreme Court, and now the Justice Dept. Most of this interest is desirable, although some activities, such as the Justice Dept. stand on AIDS and employment, may become dangerous. But the worst thing public health professions could do would be to become turf protectors. We don't have the resources to do what needs doing. We don't have the money, the people, the access, or the necessary authority. We require the help of others. By definition, public health is the health of people in the aggregate and therefore is everybody's business.

Our role will continue to be one of identifying the issues, defining and delineating the problems, and proposing reasonable solutions. We must continue to be the conscience of public health, as we work to implement programs, welcoming all the help we can get The AMA actions rejuvenate the collective spirit of all public health workers.

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Executive Privilege

Happy New Year to all of you!

Now that our political season is behind us, let us reexamine our involvement and participation. Did you hang a sign, attend a fund raiser, write a check, host a party, address envelopes, make a phone call, or like many of us, did you do absolutely nothing at all. Yet, the results of the victorious candidate's future votes will affect our pocketbooks, our lifestyles, our environment, and certainly, our future.

Physicians command respect in their communities—this respect can and should be utilized and mobilized in the political process. Times have changed. You didn't ask them to change, and medical school (and continuing medical education) never addressed the host of problems that you and organized medicine face today. But change has come—change that threatens to dissipate the quality of medical care by subjugating the physician to government and the political process. The need for physicians to respond is vital to their economic and philosophical survival. You need to recapture your humanitarian image which has eroded over the past two decades.

Although political infertility is a disease of busy lives, it is by no means fatal. But you must treat more than the symptoms—you must treat the underlying cause—apathy. This past year saw no national message or theme that captured the attention of the voters. I believe that was part of the reason for such a disappointing turnout in local and national elections. Voter apathy was apparent, even in the face of mass media overkill that attempted to sell us another story.

So what's the cure? Let's begin by asking questions and by attempting to know the persons who represent us and our interests in state and local government. Attend a political caucus, write a letter to your legislator, contribute to ALPAC. But above all, make the first critical step toward involvement.

Charles Kettering said "We should be concerned about the future, because that's where we have to spend the rest of our lives". The future of organized medicine in Alaska, the physician himself, and the community of patients he serves CAN be impacted by one step forward. As for an infant, the first one's the hardest!

Raymond Schalow
Executive Director
Alaska State Medical Association

Auxiliary News:

Alaska State Medical Association Auxiliary

Dear Doctors:

Since you will be receiving this "after the fact", I hope you enjoyed the raffle we held in November to raise $6,000 to fund the ASTHMA CHAMP CAMP next summer. As the ASMA auxiliary project, we will utilize other fund raising methods if the required sum isn't reached by the raffle. The prizes were terrific, and I hope you won some of them!

The annual September Salad Luncheon was graced by the presence of the national president of the AMA-Auxiliary, Mrs. Patricia Durham, of Houston, Texas. One of her comments reflects the theme of her focus this year and perhaps you will appreciate it as well: "I say to you, the question is not WHY belong, the question is can you afford NOT to belong? I think not. I think rather we must respect the AMA-Auxiliary as the only organization that can help physician spouses impact today's changes. We must do those things to improve the publics' image of their doctors and to make contributions to health in our communities; things the doctors don't have time to do." Her point is well taken. If these issues are not dealt with, the quality of health care in the future will not be preserved, and the public will be the greatest losers of all. Your auxiliary wishes you happy holidays!

Patrice Gerster
President, ASMA Auxiliary

Auxiliary News:

AMS Auxiliary

The Anchorage Medical Society Auxiliary was honored with a visit from our national president, Mrs. Pat Durham, in early September. Mrs. Durham held a conference on "The Value of the AMA Auxiliary in Today's Medical Climate", in which she stressed the importance of the auxiliary's activities in relationship to our spouse's profession, both now and in the future. The conference was followed by our fall membership luncheon at the home of Fran Marbarger.

October 11 was the date set for the "Totally Tasteless and Tacky Trot". An award was given for the tackiest of the tacky. Dancing, lip sync, hotdogs, and tasteless hors d'oeuvres made it an evening hard to forget. All the proceeds benefit the Children's Health Fair and the AMA Educational Relief Fund.

Also in October, the auxiliary worked with United Way, distributing pledge packages to physician offices. In November, we helped sort canned food for the Salvation Army during their program to provide holiday meals for the less fortunate.

Our annual Christmas Social held on December 12, was an opportunity to visit and relax with other auxiliary membersduring the busy holiday season.

The "Annual Medical Society Christmas Dinner" was changed for the first time to a Sunday Brunch. The Medical Society installed their 1987 officers amongst lively entertainment.

The most important news is that the Anchorage Medical Society has agreed to cosponsor, with the auxiliary, a Children's Health Fair to be held May 9. The two goals of the fair are to encourage children to live a more healthy lifestyle and to create a more positive public image of the medical community in Anchorage. You will be hearing much more about this project, and we will need everyone's help.

I encourage each new and renewing member to get involved in the auxiliary this year. We have a variety of programs to interest you. We need your talents.

Alice Samuelson
President, AMS Auxiliary

DOCTOR,
is it time for a change?

- You're spending too much time on paperwork.
- You want to live in Europe, not just vacation there for a couple of weeks.
- You want to get involved with academic medicine, full-time.
- You want to subspecialize, but can't support your family on a fellow's stipend.

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President's Page

Some of the greatest conversations happen on airplanes. On the way home from a continuing education meeting last February, I had the great pleasure of sitting next to an articulate, pleasant man in his 50's who is a senior pilot for United Airlines. We chatted for a couple of hours about the effect of deregulation on the airline's income and morale, the air traffic controllers' strike and mass firing, and the performance of the substantially new cadre of air traffic controllers that now watch the skies. We also got around to talking about pilots and doctors. (Thank heaven we didn't get on the subject of doctor-pilots!)

My new friend was telling me about some of the difficulties that pilots have reaching agreement on various issues. He noted that pilots for major airlines once commanded considerably higher salaries than are being offered now, and that their economic muscle has diminished noticeably. Yet their Association meetings are marked by strong individual wills, slow to bend for the common good. At one point he turned to me and said, "Do you guys have trouble reaching . . ." "Consensus?", I said quickly. "Yup, that's it," he said. "We sure do," I replied.

He and I have both noticed that our professions are possessed of less economic muscle than we once had or wish we had now. We agreed that both pilots and doctors are slowly learning that cooperation and the formation of consensus pays dividends, and that the economic situation in both of our fields is going to force this lesson upon us even more clearly.

Yes sir, pilots and doctors both are doing a lot more cooperating now than we have done in the past, and we have a great deal more to do in the future. I left my talk with this fine gentleman feeling not so alone in my concerns.

Donald E. Thieman, M.D.
President
Alaska State Medical Association

ASMA Convention Update

The Alaska State Medical Association's 1987 Annual Convention is planned for the Centennial Hall Convention Center in Juneau, Alaska, June 3-7th, 1987.

Plans are in progress that will, hopefully, provide over 100 exhibitors. A broad and comprehensive CME program is also being prepared. Plan to be there! See you in June!

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Attention

The Bank is now the owner and is selling 4 Medical/Dental designed offices suites at a discount. If you are in the Health Care Field, this is an opportunity that you won't want to pass up.

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Letters to the Editor . . .

Arthroscopy or Arthrogram?

To the Editor:

I have been somewhat concerned in the last year or so at the number of patients who have come seeking advice on knee difficulties with the expectation that there would be a prompt arthroscopic procedure done on their knee without other investigative work. I continue to feel that an arthrogram is an acceptable way to evaluate many meniscal and cruciate ligament lesions.

If an individual has a locked knee then an arthrogram has little to offer as a scope or open procedure is indicated. However, in instances where people have low grade knee symptoms such as intermittent aching, occasional popping without locking, or intermittent swelling, a great deal of information can often be obtained from an arthrogram.

I have read and heard criticism of that procedure implying that the chemical synovitis from the arthrogram causes more patient discomfort than an arthroscopy. In my experience, this has not been the case. In the few instances where synovitis is a difficulty, usually a non-steroidal anti-inflammatory for a few days greatly modifies the symptoms.

It is true that the diagnostic acuity of an arthroscopy exceeds that of an arthrogram. However, the cost of an arthrogram is substantially less, and I have seen far fewer complications from arthrograms over the years than from arthroscopies. In the last couple of years I have participated in the review and care of devastating arthroscopic infections which I think ought to temper our judgment in referring patients for these procedures.

I would like to emphasize that the arthrogram is a diagnostic tool available to any practitioner. There is nothing at all wrong with treating someone conservatively with physical therapy and appropriate medications in the case where the arthrogram is negative and there is no knee locking.

Arthroscopic surgery of the joints is a wonderful advance in our ability to care for orthopedic difficulties, but I think its limitations and potential complications need to be kept firmly in mind.

Sincerely,
R. W. Garner, M.D.
Anchorage, Alaska

To the Editor:

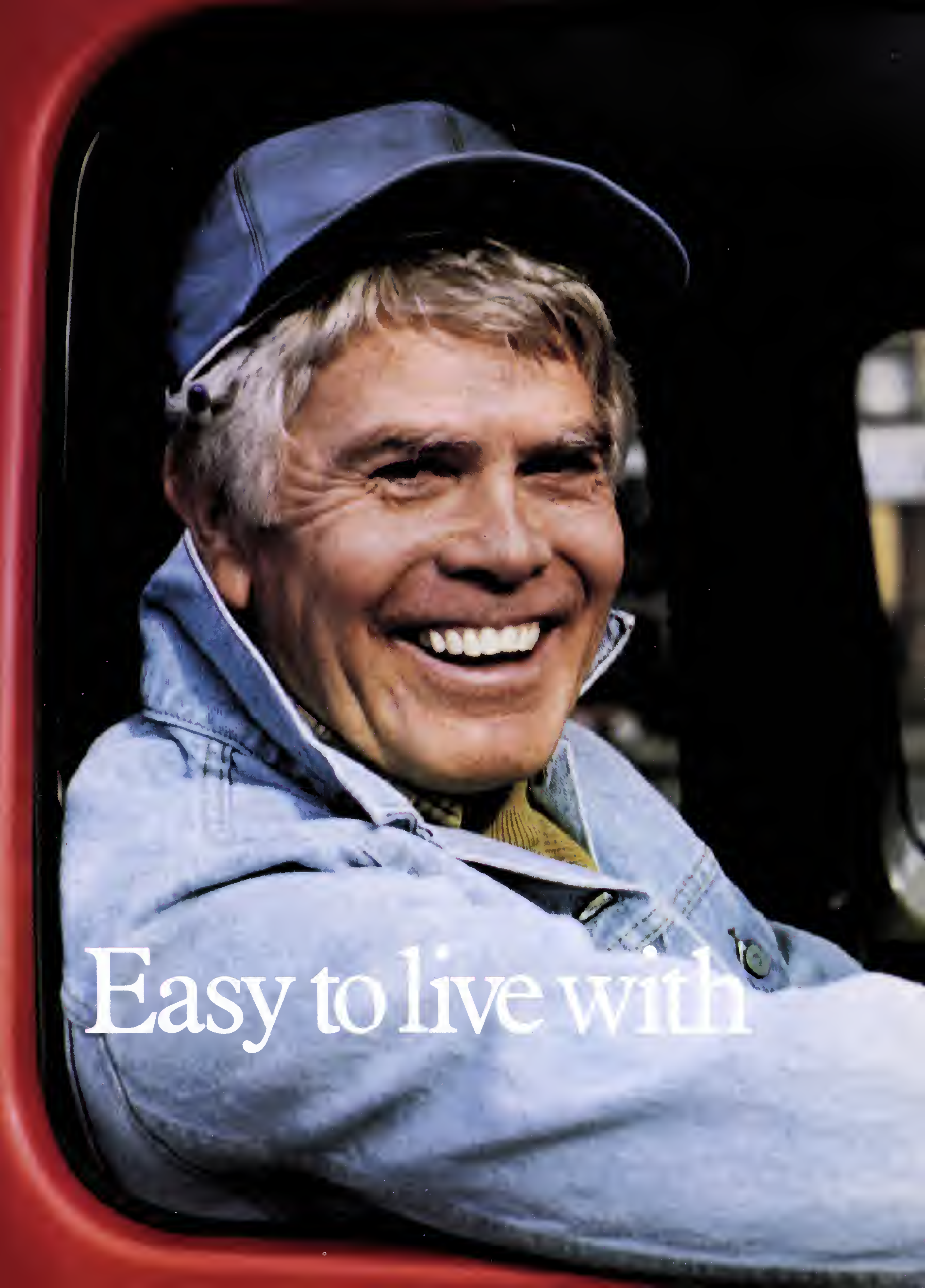
While probably just another example of one consistent mystery of human life, that human behavior tends to go in cycles, it is sadly ironic how miserable so many of us seem to be in the midst of scientific medicine's most spectacular accomplishments.

One of the reasons for this may well rest in the fact that our social and behavioral sciences are such infants. There is, I believe, a more clearly understood reason. But such understanding may be clouded by the very accomplishments of medical science. We, medicine's practitioners, and the public frequently believe our joint fantasy of nearly complete control over disease and illness. Nowhere does this fantasy belief do more harm than in the areas of cost/access in health care and accountability/malpractice for medical practice.

Both these problem areas, availability and liability, continue to be targets for our century's brightest analysts. When they write or speak, with aid from the latest computer and communications technology, an accurate, all-encompassing formula seems ever more elusive. Experienced practitioners, in all arenas of human interaction, are able to see somewhat through our fantasy clouding. We can understand that life is a gift and human friendship, especially human intimacy, is a mystery. Presuming to try to preserve life, individual, precious life daily throws us as health care practitioners directly into such mystery. We should not be too surprised that our bookkeepers and the nation's medical economists cannot put exact price tags on our services. We can continue to experience the real appreciation individual humans have for our efforts, especially when we fail despite our very best efforts. We can accept our failures, as we are now and then reminded — despite advertising and professional education arrogance — that medicine is at heart a service. Despite our failures, we can continue in the best scientific traditions to be honest with ourselves and each other by asking; "What did we do wrong?, What is wrong about our current theory?, And how can we improve this or that technique?".

In short, we can renew a desperately needed par of the mysterious cycle of human behavior. We can balance our ruinous current epidemic of fanatic belief in the "bottom line of budgets in the black" and complete material security. We can continue to find faith in the real goodness of human hearts. We can continue to trust other humans. We can appreciate the real mystery that "most of us are doing the best we can".

George W. Brown, M.D.
Palmer, Alaska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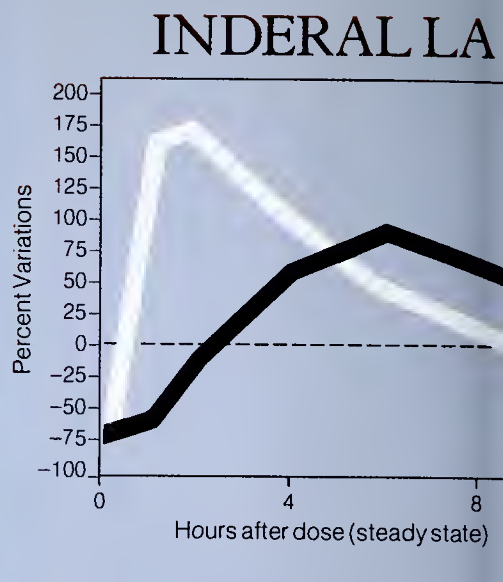

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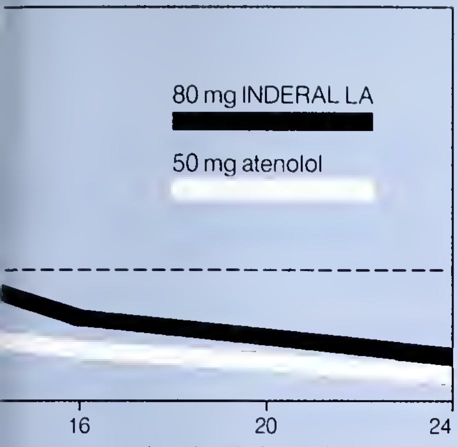

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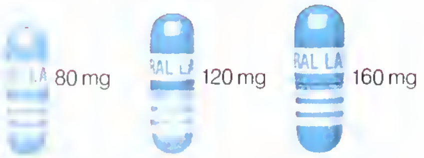

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CONTRAINDICATIONS

Propranolol hydrochloride (INDERAL® LA): Propranolol is contraindicated in. 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with propranolol.

Hydrochlorothiazide: Hydrochlorothiazide is contraindicated in patients with anuria or hypersensitivity to this or other sulfonamide-derived drugs.

WARNINGS

Propranolol hydrochloride (INDERAL® LA): CARDIAC FAILURE. Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated, and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or propranolol should be discontinued (gradually, if possible)

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction following *abrupt* discontinuance of propranolol therapy Therefore, when discontinuance of propranolol is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when propranolol is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If propranolol therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute propranolol therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD, IN GENERAL, NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution, since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure.

Hydrochlorothiazide: Thiazides should be used with caution in severe renal disease. In patients with renal disease, thiazides may precipitate azotemia In patients with impaired renal function, cumulative effects of the drug may develop.

Thiazides should also be used with caution in patients with impaired hepatic function or progressive liver disease, since minor alterations of fluid and electrolyte balance may precipitate hepatic coma

Thiazides may add to or potentiate the action of other antihypertensive drugs Potentiation occurs with ganglionic or peripheral adrenergic-blocking drugs.

Sensitivity reactions may occur in patients with a history of allergy or bronchial asthma

The possibility of exacerbation or activation of systemic lupus erythematosus has been reported

PRECAUTIONS

Propranolol hydrochloride (INDERAL® LA): GENERAL. Propranolol should be used with caution in patients with impaired hepatic or renal function. Propranolol is not indicated for the treatment of hypertensive emergencies

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that propranolol may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure

CLINICAL LABORATORY TESTS. Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs, such as reserpine should be closely observed if propranolol is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity, which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies, in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

PREGNANCY Pregnancy Category C. Propranolol has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximal recommended human dose. There are no adequate and well-controlled studies in pregnant women. Propranolol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

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NURSING MOTHERS Propranolol is excreted in human milk. Caution should be exercised wh propranolol is administered to a nursing mother.

PEDIATRIC USE. Safety and effectiveness in children have not been established

Hydrochlorothiazide: GENERAL. Periodic determination of serum electrolytes to dete possible electrolyte imbalance should be performed at appropriate intervals.

All patients receiving thiazide therapy should be observed for clinical signs of fluid or electroly imbalance, namely Hyponatremia, hypochloremic alkalosis, and hypokalemia. Serum and urir electrolyte determinations are particularly important when the patient is vomiting excessively receiving parenteral fluids Medication such as digitalis may also influence serum electrolyte Warning signs irrespective of cause are. Dryness of mouth, thirst, weakness, lethargy, drowsines restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, ar gastrointestinal disturbances such as nausea and vomiting

Hypokalemia may develop, especially with brisk diuresis, when severe cirrhosis is present, during concomitant use of corticosteroids or ACTH

Interference with adequate oral electrolyte intake will also contribute to hypokalemia Hyp kalemia can sensitize or exaggerate the response of the heart to the toxic effect of digita (eg, increased ventricular irritability). Hypokalemia may be avoided or treated by use of potassiu supplements, such as foods with a high potassium content.

Any chloride deficit is generally mild and usually does not require specific treatment, exce under extraordinary circumstances (as in liver or renal disease). Dilutional hyponatremia may occ in edematous patients in hot weather, appropriate therapy is water restriction, rather than admini tration of salt, except in rare instances when the hyponatremia is life-threatening. In actual sa depletion, appropriate replacement is the therapy of choice

Hyperuricemia may occur or frank gout may be precipitated in certain patients receiving thiazid therapy.

Insulin requirements in diabetic patients may be increased, decreased, or unchanged. Diabete mellitus which has been latent may become manifest during thiazide administration.

If progressive renal impairment becomes evident, consider withholding or discontinuing diuret therapy.

Thiazides may decrease serum PBI levels without signs of thyroid disturbance.

Calcium excretion is decreased by thiazides. Pathologic changes in the parathyroid gland wi hypercalcemia and hypophosphatemia have been observed in a few patients on prolonge thiazide therapy The common complications of hyperparathyroidism, such as renal lithiasis, bor resorption, and peptic ulceration, have not been seen. Thiazides should be discontinued befo carrying out tests for parathyroid function

DRUG INTERACTIONS: Thiazide drugs may increase the responsiveness to tubocurarine.

The antihypertensive effects of thiazides may be enhanced in the postsympathectomy patier Thiazides may decrease arterial responsiveness to norepinephrine. This diminution is not sufficie to preclude effectiveness of the pressor agent for therapeutic use.

PREGNANCY Pregnancy Category C. Thiazides cross the placental barrier and appear in co blood The use of thiazides in pregnancy requires that the anticipated benefit be weighed again possible hazards to the fetus. These hazards include fetal or neonatal jaundice, thrombocytopeni and possibly other adverse reactions which have occurred in the adult

NURSING MOTHERS Thiazides appear in human milk. If use of the drug is deemed essentia the patient should stop nursing

PEDIATRIC USE. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Propranolol hydrochloride (INDERAL® LA): Most adverse effects have been mild ar transient and have rarely required the withdrawal of therapy

Cardiovascular Bradycardia, congestive heart failure, intensification of AV block; hypotensio paresthesia of hands, thrombocytopenic purpura; arterial insufficiency, usually of the Raynau type.

Central Nervous System: Lightheadedness; mental depression manifested by insomni lassitude, weakness, fatigue, reversible mental depression progressing to catatonia; visu disturbances, hallucinations; an acute reversible syndrome characterized by disorientation f time and place, short-term memory loss; emotional lability; slightly clouded sensorium; ar decreased performance on neuropsychometrics.

Gastrointestinal Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, cons pation; mesenteric arterial thrombosis; ischemic colitis.

Allergic: Pharyngitis and agranulocytosis; erythematous rash; fever combined with aching ar sore throat, laryngospasm and respiratory distress.

Respiratory Bronchospasm

Hematologic: Agranulocytosis; nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune. In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous Alopecia, LE-like reactions, psoriasiform rashes; dry eyes; male impotence; ar Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the ski serous membranes, and conjunctivae reported for a beta blocker (practolol) have not bee associated with propranolol.

Hydrochlorothiazide:

Gastrointestinal Anorexia, gastric irritation, nausea, vomiting, cramping, diarrhea, constipatio jaundice (intrahepatic cholestatic jaundice); pancreatitis, sialadenitis.

Central Nervous System Dizziness, vertigo; paresthesias; headache; xanthopsia.

Hematologic: Leukopenia; agranulocytosis; thrombocytopenia; aplastic anemia

Cardiovascular: Orthostatic hypotension (may be aggravated by alcohol, barbiturates, narcotics)

Hypersensitivity Purpura, photosensitivity; rash; urticaria; necrotizing angiitis (vasculiti cutaneous vasculitis); fever; respiratory distress, including pneumonitis, anaphylactic reactions

Other: Hyperglycemia, glycosuria; hyperuricemia, muscle spasm; weakness; restlessnes transient blurred vision.

Whenever adverse reactions are moderate or severe, thiazide dosage should be reduced therapy withdrawn.

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REFERENCES

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Please see following page for brief summary of prescribing information.

For moderate-to-severe vasomotor symptoms

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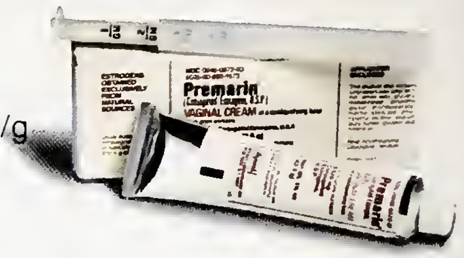

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream in a nonliquefying base

1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA

Three independent case control studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade The three case control studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13.9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible. When prolonged treatment is medically indicated, the patient should be reassessed on at least a semiannual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equiestrogenic doses
2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.
The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a non-steroidal estrogen, have an increased risk of developing in later life a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix. Although these changes are histologically benign, it is not known whether they are precursors of malignancy Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb reduction defects. One case control study estimated a 4.7-fold increased risk of limb reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb reduction defects in exposed fetuses is somewhat less than 1 per 1,000 In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine. It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0.3 mg, 0 625 mg, 0.9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens. Cream is available as 0.625 mg conjugated estrogens per gram.
INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) **Osteoporosis (abnormally low bone mass).** Atrophic vaginitis Kraurosis vulvae. Female castration.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae. PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).
Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia. Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established. There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens (See PRECAUTIONS.) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects.
CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions: 1 Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2. Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (See Boxed Warning). 4. Undiagnosed abnormal genital bleeding. 5. Active thrombophlebitis or thromboembolic disorders. 6. A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).
WARNINGS: Long-term continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver There are now reports that estrogens increase the risk of carcinoma of the endometrium in humans. (See Boxed Warning.) At the present time there is no satisfactory evidence that estrogens given to postmenopausal women increase the risk of cancer of the breast, although a recent study has raised this possibility. There is a need for caution in prescribing estrogens for women with a strong family history of breast cancer or who have breast nodules, fibrocystic disease, or abnormal mammograms. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement; it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction. Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users. An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives. If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization. Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism and thrombophlebitis. When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk.

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock. Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be carefully observed. Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases.
PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc. Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed. Preexisting uterine leiomyomata may increase in size during estrogen use. The pathologist should be advised of estrogen therapy when relevant specimens are submitted If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated. Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not complete. If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a Increased sulfobromophthalein retention.

b. Increased prothrombin and factors VII, VIII, IX, and X; decreased antithrombin 3; increased norepinephrine-induced platelet aggregability.

c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T4 by column, or T4 by radioimmunoassay. Free T3 resin uptake is decreased, reflecting the elevated TBG; free T4 concentration is unaltered.

d. Impaired glucose tolerance.

e. Decreased pregnanediol excretion

f. Reduced response to metyrapone test.

g. Reduced serum folate concentration.

h. Increased serum triglyceride and phospholipid concentration. As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk
ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow; dysmenorrhea; premenstrual-like syndrome; amenorrhea during and after treatment; increase in size of uterine fibromyomata; vaginal candidiasis, change in cervical erosion and in degree of cervical secretion; cystitis-like syndrome; tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued; erythema multiforme; erythema nodosum; hemorrhagic eruption; loss of scalp hair; hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea; increase or decrease in weight; reduced carbohydrate tolerance; aggravation of porphyria; edema; changes in libido
ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females.
DOSAGE AND ADMINISTRATION:
PREMARIN® Brand of conjugated estrogens tablets, USP
1. *Given cyclically for short-term* use *only.* For treatment of moderate to severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible. Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals.
2. *Given cyclically:* Female castration. Osteoporosis. Female castration—1.25 mg daily, cyclically. Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control. Osteoporosis —0.625 mg daily. Administration should be cyclic (eg, three weeks on and one week off)

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.
PREMARIN® Brand of conjugated estrogens Vaginal Cream
Given cyclically for short-term use *only.* For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three-to-six month intervals.

Usual dosage range: 2 to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.
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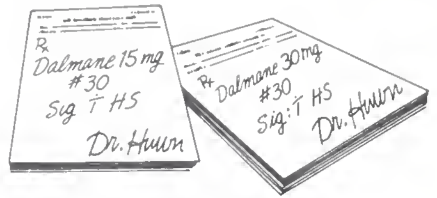

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase, and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults*: 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients*: 15 mg recommended initially until response is determined.

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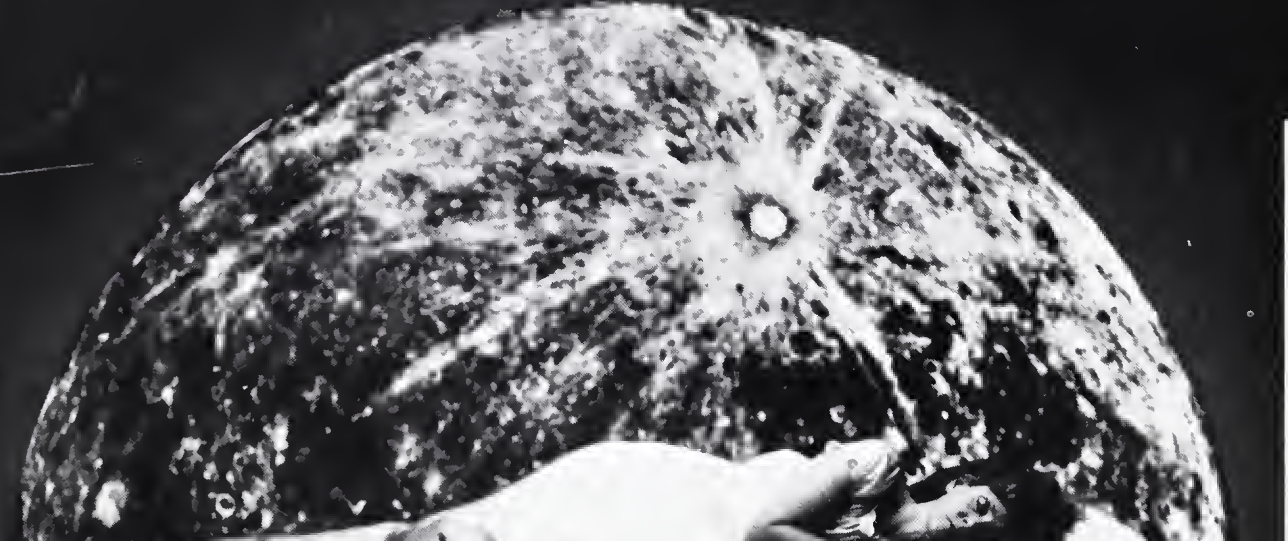

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